Society of Cardiovascular Computed Tomography / North American Society of Cardiovascular Imaging – Expert Consensus Document on Coronary CT Imaging of Atherosclerotic Plaque

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Abstract
Coronary computed tomographic angiography (CCTA) provides a wealth of clinically meaningful information beyond anatomic stenosis alone, including the presence or absence of nonobstructive atherosclerosis and high-risk plaque features as precursors for incident coronary events. There is, however, no uniform agreement on how to identify and quantify these features or their use in evidence-based clinical decision-making. This statement from the Society of Cardiovascular Computed Tomography and North American Society of Cardiovascular Imaging addresses this gap and provides a comprehensive review of the available evidence on imaging of coronary atherosclerosis. In this statement, we provide standardized definitions for high-risk plaque (HRP) features and distill the evidence on the effectiveness of risk stratification into usable practice points. This statement outlines how this information should be communicated to referring physicians and patients by identifying critical elements to include in a structured CCTA report - the presence and severity of atherosclerotic plaque (descriptive statements, CAD-RADS™ categories), the segment involvement score, HRP features (e.g., low attenuation plaque, positive remodeling), and the coronary artery calcium score (when performed). Rigorous documentation of atherosclerosis on CCTA provides a vital opportunity to make recommendations for preventive care and to initiate and guide an effective care strategy for at-risk patients.

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The traditional approach to imaging of coronary anatomy with coronary computed tomographic angiography (CCTA) has been oriented toward the detection of obstructive coronary artery disease (CAD). Over the past decade, evidence has accumulated concerning the prognostic significance of nonobstructive and obstructive atherosclerotic plaque including specific features that are associated with an elevated risk of major adverse CAD events (MACE). This evidence now supports that imaging of atherosclerotic plaque is important for estimating patient risk and guiding preventive care. This link between initiation and intensification of prevention with atherosclerotic imaging using CCTA is vital to improving patient outcome. In this statement from the Society of Cardiovascular CT (SCCT) and North American Society of Cardiovascular Imaging (NASCI), we provide a synthesis of evidence concerning CCTA imaging of atherosclerosis and propose data elements that radiologists and cardiologists can integrate into their CCTA interpretation. One vital part of this statement is to acknowledge the advantages and limitations of current methods used to assess atherosclerotic plaque and to identify future research in this area. In this SCCT / NASCI statement, we enlist thought leaders in the field of CCTA including experts from the field of radiology and cardiology who bring unparalleled experience to this subject of atherosclerotic imaging. This field is rapidly evolving and further advancements, including fully automated quantification of atherosclerotic plaque will necessitate future updates to this statement.

This statement focuses on the following principal issues that will influence how we think about imaging coronary atherosclerotic plaque:

1. **CT Imaging of Atherosclerotic Plaque**: Risk prediction in stable and unstable chest pain syndromes
2. **Evidence Gaps in Coronary Atherosclerotic Plaque Imaging and Future Research Needs**
3. **CT Plaque Imaging to Direct Risk-Reducing Preventive or Disease-Modifying Therapies**: Observational evidence on CCTA-guided preventive strategies
4. Minimum Data Elements for Structured Reporting For Coronary Atherosclerotic Plaque

5. Consensus Summary and Recommendations

**Coronary Atherosclerosis is the Primary Disease Process**

Imaging has traditionally focused on detecting flow limiting stenosis or its surrogates. Detection of the presence and severity of obstructive CAD by CCTA guide clinical decision making for secondary prevention including consideration of coronary revascularization. However, atherosclerosis is the primary disease process that mediates risk and outcome and stenosis is just one of its many subsets. Importantly, for risk assessment, patients with more extensive, multivessel CAD are at highest risk while those without any plaque or stenosis comprise those at lowest risk (**Central Illustration**). This document focuses on the presence and extent of atherosclerotic plaque that elevates risk above that of patients without any documented plaque. This ranking of disease presence and severity remains vital to providing a comprehensive assessment of patient risk.

The evolving paradigm for CCTA imaging represented in this document expands this viewpoint beyond detecting obstructive lesions to imaging coronary vessel pathology itself: characterizing and providing measurement of the burden of atherosclerosis, identifying how its composition influences risk and outcomes, and potentially modifying risk with initiation or intensification of therapy. We propose that imaging of atherosclerotic plaque in order to target effective preventive care strategies reinforces the concept that early detection and intervention has the greatest potential for improved event-free life years for at-risk patients. However, current randomized trials are lacking and do not proscribe a detailed evaluation and treatment pathway. In this statement, we synthesize the observational evidence and patterns as they unfold about the impact of treatment on atherosclerotic plaque.

**Targeting the Appropriate Patient Population**

This guideline will focus discussion on the use of imaging of atherosclerosis with CCTA for symptomatic patients. Although evidence does exist with regards to the role of imaging in
asymptomatic individuals, there are separate guidance documents from SCCT on the clinical indications for coronary artery calcium (CAC) scoring in asymptomatic patients\(^1\) and the CAC data and reporting system statement (CAC-DRS\(^{TM}\)).\(^2\)

**Methodologic Considerations in Atherosclerotic Plaque Imaging**

This document will highlight evidence on both qualitative as well as quantitative atherosclerotic plaque assessment. However, at present, in the absence of validated tools for quantitative assessment, we recommend integration only of semi-quantitative measures of atherosclerotic plaque into clinical reporting and propose data elements that radiologists and cardiologists can integrate into their CCTA interpretations today. While they remain incompletely defined at present, the field is evolving rapidly. The current quality standards for evidence are detailed in Table I.

Provided limitations in spatial resolution and image quality, plaque analysis should be restricted to epicardial vessels 2.0 mm in size and greater. Moreover, prior SCCT guidelines make recommendations for initial setting the window width and window levels (initial window width: 800 Hounsfield Unites [HU] and level: 300 HU) for accurate gray-scale differentiation between vessel wall and calcified and noncalcified plaque.\(^3,4\) More recently, reports have applied varied approaches including: a) initial settings starting at a window width of 700 HU and window level of 200 HU; with subsequent changes determined by the imager,\(^5\) b) employing a fixed HU setting of 740/220, including a fixed 0.3 mm gap between vessel wall and lumen,\(^6\) and c) vessel wall thresholds adjusted to the lumen using a 155%/65% window width/level of the luminal intensity.\(^5,7\) Plaque analysis has been validated across recommended target heart rate (60-80 beats per minute) and standard tube voltage (100-120 kV).\(^8\) Whether or not to employ low kVp imaging is guided by the specific clinical question being evaluated as well as whether radiation saving techniques are desirable (e.g., young age). Lower tube voltage would impact HU ranges for plaque measurements.

**Importance of Nonobstructive CAD and CAD Event Risk**
An emerging message from the published literature, over the past decade, is that patients with nonobstructive CAD (i.e., 1-49% stenosis) have worse prognosis when compared to those without any stenosis or plaque (i.e., normal CCTA). Figure 1 provides a synthesis of available evidence, noting that nearly 1 in 3 patients referred for CCTA for suspected CAD will have nonobstructive CAD.\textsuperscript{9-14,3, 15-23} One-third of patients referred to CCTA have nonobstructive CAD while ~10-15% of patients have obstructive CAD.\textsuperscript{24} The annualized event rate for patients with nonobstructive CAD is ~1.6% as compared to 0.2% for those with a normal CCTA. The adjusted hazard for MACE for nonobstructive CAD as compared to a normal CCTA ranged from 1.5 to 7.2; when controlling for CAD risk factors or a risk score.\textsuperscript{9-14,3, 15-23}

Thus, a first step for all CT imagers to comprehend that identification of nonobstructive plaque is an important aspect of identifying at-risk patients. One commonly reported measure is the semi-quantitative assessment of the number of coronary segments with plaque, independent of the stenosis severity, using the segment involvement score (SIS).\textsuperscript{25} The SIS provides an assessment of the extent of atherosclerosis and has higher reproducibility than descriptive terms. An SIS score $>5$ has been associated with an elevated event risk ($p<0.0001$).\textsuperscript{26}

**High Risk Plaque (HRP) Features on CCTA**

Histological assessment of coronary atherosclerosis has identified plaque features found in culprit lesions of patients who succumbed to sudden cardiac death, in particular, thin cap fibroatheroma (TCFA).\textsuperscript{27-30} Beyond the thin cap, other features comprising the TCFA are large plaque volume and necrotic core. Some of these features have been identified using invasive imaging methods, including IVUS,\textsuperscript{31} optical coherence tomography (OCT),\textsuperscript{32} and near-infrared spectroscopy in patients with acute coronary syndromes. Each of these methods has its own limitations in providing a comprehensive picture of the vessel wall, extent of atheroma burden across the entire tree and stenosis.\textsuperscript{33} Figure 2 depicts examples of stable and high-risk plaque pathology as characterized by IVUS and OCT.
With the exception of the thin cap, these features of TCFA (i.e., the presence of a large lipid core, positive remodeling [PR], and large plaque burden) can also be identified with CCTA with the advantage that it is noninvasive. CCTA can characterize and identify abnormalities in the coronary artery wall itself across the entire coronary tree, rather than just the presence of luminal stenosis. This includes the identification of both calcified and noncalcified plaque, and further classification of plaque size and specific HRP features. These HRP features found on CCTA correlate with histological and invasive assessment of plaque vulnerability, and are more frequent in patients with an acute coronary syndrome (ACS) at the time of CCTA or for those who subsequently develop ACS or other major adverse CAD events (MACE) after CCTA.

**Figure 3** details the hierarchical importance of morphological characteristics of plaque vulnerability.

**HU Density and Plaque Composition**

The composition of atherosclerotic plaque is categorized using HU density with lower to higher values correlating to necrotic core, fibro-fatty, fibrous, and calcified plaque by intravascular ultrasound and histopathology. Several interpretive platforms are available that employ slightly different HU density ranges for categorization. The composition of plaque is categorized as follows: a) dense calcium for HU densities >350’ b) fibrous plaque for a range of values from 131 to 350 HU; c) fibro-fatty plaque for a range of values from 31 to 130 HU; and necrotic core for a range of values from -30 to 30 HU. **Figure 4** provides an example of CCTA characterization of plaque composition. Importantly, HU densities on CT overlap across compositional subgroups on IVUS. Normalization of the luminal contrast attenuation (plaque / lumen HU ratio) or adaptive thresholds can improve categorization of lipid-rich versus fibrous plaque. Most of the software programs have been correlated with IVUS measurements, but data is not available directly comparing the accuracy of the varied programs. As such, CT imagers should realize that variations in plaque measurements may result from different
programs. Figure 5 includes the results from several published reports detailing CCTA-verified plaque composition and clinical outcomes.38, 39

**HRP and Culprit ACS Lesions**

Several CCTA characteristics associated with these histological and invasive features of plaque vulnerability40 were confirmed as plaque features associated with culprit ACS lesions.27, 31-33, 40, 41 The presence of HRP features on CCTA are common in culprit lesions of ACS.27, 31, 33, 40-43 The major plaque characteristics included in HRP definitions are low attenuation plaque (LAP), and positive remodeling (PR) and have conveniently been termed as 2-feature positive plaques.38, 44, 45 PR is the presence of an outer vessel diameter which is ≥10% greater than the mean of the diameter of the normal adjoining segments, also labelled as the remodeling index (RI) >1.1.34, 46 Measurement of an RI >1.1 can be challenging in smaller diameter vessels where limitations in spatial resolution may impair detection. LAP is conventionally defined as the presence of a central focal area within the plaque which has a low CT attenuation which is usually defined as at least 1 voxel with <30 HU;47, 48 although other thresholds (e.g., <60 HU and <90HU) have also been used.49-51 This definition based on a single voxel threshold can be impacted by image noise. Other less studied features include the napkin-ring sign (NRS) which suggests the presence of circumferential necrotic core33, 41, 52 and spotty calcification (SC) as compared to more dense and plate-like calcification.53 The NRS is described as a central area of low CT attenuation that abuts the lumen and a ring like higher attenuation plaque tissue surrounds this low attenuation area.27, 33, 40, 54-56 A NRS is associated with advanced atherosclerotic lesions by histology, including a large lipid core or thrombus. Several definitions of SC are used in the literature, the most commonly applied is the presence of small focal calcifications <3 mm diameter in any direction. Another definition includes calcium length (in the longitudinal direction of the vessel) <1.5 times the vessel diameter and width (calcification extent perpendicular to the longitudinal direction of the vessel) <2/3 of the vessel diameter.32, 57,
These HRP features are easy to identify when combined with simple manual measurement (e.g., distance, region of interest) on standard workstations.

In a secondary analysis from the ROMICAT (Rule Out Myocardial Infarction using Computer Assisted Tomography) II trial, the presence of HRP was associated with an increased risk of ACS during the index hospitalization; independent of stenosis severity. A meta-analysis of 18 smaller studies confirm the association between HRP features and the culprit ACS lesion.

The CAD-RADS™ classification defines HRP as the presence of a minimum of two of the four HRP features in the same coronary plaque. Although ACS risk increases when two or more HRP features are present, any of the four measures correlate with event risk. However, some studies have not included SC in the definition of HRP and others have suggested that the presence of SC was not associated with an elevated risk when compared to high risk, LAP and the NRS.

Prognostic value of HRP in Patients with Stable Chest Pain

HRP is a frequent finding on CCTA, occurring in 15-34% of patients with stable chest pain. Studies of symptomatic patients followed after CCTA have shown that the presence of HRP was associated with an increased risk of subsequent MACE. In a large study of 1,059 patients followed for a mean of 27 months, the presence of both PR and LAP was associated with ~22-fold increase in ACS risk. In a larger study of 3,158 patients followed for ~4 years, Motoyama et al. reported that the presence of HRP (i.e., PR and LAP) was associated with 8-fold increase of subsequent ACS. Several other studies as well as a meta-analysis found similar results from a large number of patients undergoing CCTA for stable chest pain.

Recently, investigators in the PROMISE (Prospective Multicenter Imaging Study for Evaluation of Chest Pain) trial performed an assessment of HRP (defined as the presence of PR, LAP, or the NRS) in patients randomized to CCTA. The presence of HRP was associated with a ~70% increased risk of MACE (death, myocardial infarction [MI], or hospitalization for
unstable angina) at ~2 years of follow-up. The predictive value of HRP was strongest in younger patients, women, and in those with nonobstructive CAD.

In the SCOT-HEART (Scottish Computed Tomography of the HEART) trial, the presence of HRP (defined as the presence of PR and/or LAP) was associated with an increased risk of coronary heart disease (CHD) death or MI at 5 years.65 Interestingly, HRP did not retain independent prognostic value when the CAC score was included as a covariate. The lack of improvement in risk prediction by HRP is likely the result of the CAC score as a measure of overall coronary plaque burden, while the HRP assesses the specific features of individual plaques. This secondary analysis reveals an important consideration that HRP are but one of many risk markers and that the overall plaque burden, as measured by CAC, provide additional information to guide risk stratification.

Challenges with prognostication using HRP including a high prevalence (e.g., in the PROMISE trial, HRP identified in 40% of patients with nonobstructive plaque and 75% of patients with obstructive CAD) and a reduced positive predictive value; similar to invasive testing for identification of HRP. In part, the overall low risk observed in most studies is plausible given that plaque characteristics are dynamic and can change over time, particularly following initiation or intensification of medical therapies.65 It appears that the presence of HRP may best predict near as compared to long-term risk.65

**Quantitative Measurement of Atherosclerotic Plaque**

Technological developments in CCTA now allow semi-automated/automated software solutions for quantitative assessment of atherosclerotic plaque characteristics.57-60, 62, 63, 67-87 Currently available semi-automated plaque assessment is time consuming, and fully automated quantification of atherosclerotic plaque based largely on artificial intelligence methods for image segmentation may be available in the near future for investigational purposes. **Figure 6** illustrates an example of the quantitative measurement of atherosclerotic plaque, along with a
Forrest plot of the results of studies correlating CCTA and IVUS measurements of plaque volume.\textsuperscript{74}

**Quantitative Plaque Assessment and Prognostic Value for Major CAD Events**

Several studies have shown the feasibility of quantitative assessment of plaque and established plaque volume as a predictor of future CAD events.\textsuperscript{72, 88-90} In acute chest pain populations, quantitative plaque assessment was associated with ACS and culprit plaques in ROMICAT I and II.\textsuperscript{73, 91} Furthermore, quantitative plaque assessment improved definitions of HRP features.\textsuperscript{27} Patients with acute chest pain diagnosed with ACS had increased atherosclerotic plaque volume, notably fibro-fatty and necrotic core plaque, when compared to those with stable disease.\textsuperscript{92}

The ICONIC (Incident COroNary Syndromes Identified by Computed Tomography) case-control series is the largest (n=234 matched case and control pairs) to evaluate quantitative CCTA precursors of ACS. In ICONIC, both HRP features and quantitative assessment of fibrofatty and necrotic core plaque volume was more often associated with patient experiencing ACS; independent of coronary stenosis.\textsuperscript{36} HRP and quantitative LAP and SC were also predictors of future ACS culprit lesions in a population of patients with diabetes mellitus.\textsuperscript{93} Feuchtner et al. observed that LAP (<60 HU) and the NRS were the strongest predictors of MACE in a follow-up study of 1,469 patients.\textsuperscript{63} Similarly, Nadjiri et al. observed similar results, while LAP, NRS, and total plaque volume were all predictive of MACE; LAP had prognostic information beyond traditional assessment of coronary stenosis.\textsuperscript{77}

Similarly, from the SCOT-HEART trial, quantitative analysis revealed that low-attenuation plaque burden (% plaque to vessel volume) was the single greatest predictor of incident MI.\textsuperscript{39} Moreover, the presence of low attenuation plaque burden >4% was associated with a nearly 5-fold increase in the hazard for MI (Figure 5). Among patients with nonobstructive CAD, a low attenuation plaque burden >4% was significantly predictive of
incident MI but no increase in risk was noted for those with nonobstructive CAD and a low attenuation plaque burden <4% (p=0.81).

Summary Statement on Risk Assessment with CCTA Findings of HRP

1. HRP characteristics are associated with ACS and future MACE. These lesions with HRP may be viewed as precursors to ACS, with the CCTA findings consistent with the invasive literature.

2. Reproducibility and Interobserver agreement for measurement of HRP requires additional investigation and improvement. Interobserver agreement for HRP remains moderate, with kappa values from 0.56 to 0.69 in research studies. This is suboptimal at present when compared to the detection of a high-grade stenosis or the presence of any atherosclerotic plaque (yes/no) or characterization as calcified, noncalcified, and partially calcified.

3. There are differences in inter-observer variability in the visual identification of individual HRP features, with PR have the best overall agreement.

4. Automated quantification of atherosclerotic plaque is available but, at this time, is investigational.

5. Importantly, although we can risk stratify with HRP, directed interventions are not yet defined. Moreover, additional data are required as the positive predictive value for HRP is variable when compared to comprehensive plaque burden measures, such as CAC.

6. HRP is a dynamic process and likely to change over time– newer HRP may appear or the existing HRP could heal in response to lipid-lowering therapy without reaching a clinical threshold of disruption. Thus, routine use in clinical practice, especially in the presence of easy to quantify technology in future, will have to await robust clinical outcome data.

Minimum Data Elements for Structured Reporting of Coronary Atherosclerotic Plaque

Coronary Plaque in Structured CCTA Reports
Standardized reporting of CCTA findings is recommended by guidelines from SCCT.\textsuperscript{2, 96, 97} The use of a structured report facilitates communication of important test results to referring physicians.\textsuperscript{98} Current guidelines focus on reporting the severity, location, and extent of stenosis and obstructive CAD. There is growing evidence of the importance of nonobstructive CAD\textsuperscript{14, 25, 99-101} and these results support the inclusion of atherosclerotic plaque assessment in structured reports for CCTA. The summary statement should include whether coronary plaque is present. Location of plaque and its extent might add value for the clinician considering further risk stratification. Further specification of the plaque type, including calcified plaque, non-calcified plaque and partially calcified plaque and features of HRP can also be included. The CAD-RADS\textsuperscript{TM} system for reporting of CCTA includes all of this information - the presence of nonobstructive plaque (CAD-RADS\textsuperscript{TM} 0: no CAD and no atherosclerosis vs. CAD-RADS\textsuperscript{TM} 1-5: coronary plaque is present) - and is a good foundation for the structured reporting elements. Inclusion of CAD-RADS\textsuperscript{TM} categories in the report is recommended in addition to verbal description of the presence of coronary plaque.

**Quantification of CAC in Structured CCTA Reports**

Unlike plaque quantification with CCTA, the quantification of total CAC score is simple and well standardized.\textsuperscript{2, 102} SCCT guidelines have indicated that CAC scanning is optional at the time of CCTA.\textsuperscript{4} For patients who undergo a non-contrast scan for calcium quantification as part of their CCTA examination, we recommend reporting the total CAC score (i.e., the Agatston score).\textsuperscript{103} The CAC score is an established marker of overall coronary plaque burden, not captured in visual CCTA assessment, and a strong predictor of future CV events.\textsuperscript{11, 104-107} When CAC scoring was performed, the summary statement of the report should state total CAC score including risk categories (0: no CAC, 1-99: mild CAC, 100-299: moderate CAC, \( \geq \)300: severe CAC). The optional category of minimal CAC (1-9) is also in common use. American College of Cardiology / American Heart Association preventive treatment guidelines are endorsed by SCCT for each gradation of CAC.\textsuperscript{108}
**Semi-Quantitative Measurement of Atherosclerotic Plaque on CCTA**

Compared to the CAC score, a drawback of reporting of visual assessment of coronary plaque on contrast-enhanced CCTA is that it does not easily quantify the overall plaque burden. Descriptive assessment (e.g., mild, moderate, and severe coronary plaque) is used in structured reports. However, as visual interpretation is subjective, the reproducibility of this approach is suboptimal, and the clinical implications of this variability is unclear. The body of the report can include description of plaque presence and plaque type at a segmental or vessel level a statement regarding the overall amount of plaque in the conclusion of the report. This can be based on a visual assessment (acknowledging the aforementioned limitations); the CAC score (if performed); or a semi-quantitative assessment of the number of coronary segments with plaque using the segment involvement score (SIS).²⁵

Prior studies have shown that each additional coronary segment with plaque is associated with ~20% increase of the risk of future CV events.²⁵, ¹⁰⁹ Bittencourt *et al.* has shown that patients with nonobstructive plaque, with a SIS ≥4, have a similar rate of CV death or MI as patients with an obstructive stenosis.⁹⁹ The SIS provides independent prognostic information above and beyond the presence of obstructive CAD. Nomograms for the SIS derived from CONFIRM registry are available and higher SIS values correlate with higher MACE risk (Figure 7).¹¹⁰

Advances in CCTA quality and technological developments in semi-automated/automated software solutions have the potential to provide clinical quantitative assessment of plaque volumes. As noted above, quantitative plaque volume has predictive value for estimating future CV events.³⁷, ⁴⁹, ¹¹¹-¹¹³ The disadvantages of quantitative plaque assessment include relatively long post-processing times, particularly in patients with extensive plaques, and variations among software platforms regarding specific plaque measurements. Thus, these measurements are currently not recommended for routine use in clinical practice. In the future, machine
Learning methods may make comprehensive quantitative assessment of coronary plaque part of standard structured reporting.

**Reporting of HRP in Structured Reporting**

The presence of HRP is an important predictor of acute and future CAD events and noted in the structured report. When present, HRP should be included in the description of a lesion (using CAD-RADS™ modifier “V”), and the report conclusion noting that the presence of HRP has been associated with a higher risk of ACS (when evaluating patients with acute chest pain) or with a higher risk of future MACE (when evaluating patients with stable chest pain).

**Recommendations for the assessment and reporting of HRP in clinical practice**

The presence of HRP should be included in CCTA reports, as is recommended in CAD-RADS™ as part of the report summary.

Currently, individual HRP features considered in this definition include all features (i.e., PR, LAP, NRS, and SC). The presence of HRP should be reported if at least two features are present in the same plaque with addition of “V” to numerical CAD-RADS™ category of 1-5. The presence of at least 2 HRP features increases the strength of association with future CAD events. In addition, when significant, the degree of PR may also be described. The presence of individual HRP features at vessel or segment level can be considered for the body of the report.

**Communicating Imaging Findings in the Context of Shared Decision Making**

It is of paramount importance that information on the presence, severity, and extent of coronary atherosclerotic plaque form a core part of shared decision making with patients after CCTA. The discussion of the results of the CCTA should include a clear description of the role of the imaging for guiding clinical management and the importance of patient adherence to prescribed therapeutic strategies of care.

The absence of coronary atherosclerosis on CCTA portends an excellent CV prognosis with very low event rates over a short to intermediate term follow-up. Patients with no evidence of atherosclerosis should be re-assured and discussions can occur regarding a de-escalation of
preventive therapies (i.e., statin); although, randomized clinical trial data supporting this strategy are not yet available. Observational data suggest that absence of coronary atherosclerosis (with a CAC score of 0) is associated with a low rate of MACE with potentially no-minimal risk-reducing benefit observed from statin therapy.\textsuperscript{114}

**Limitations of Atherosclerosis Imaging by CCTA**

The evaluation of coronary atherosclerosis is limited by the intrinsic spatial resolution of \(~0.5\) mm that can be achieved with current generation CT scanners. Atherosclerotic lesions have a thickness of 0.5-1 mm to be detected by CCTA.\textsuperscript{115} Furthermore, detection of noncalcified plaque is limited by low attenuation differences between plaque tissue and perivascular fat. Therefore, good image quality is necessary for detection of coronary plaque, with a lower level of image noise and extent of artifacts for plaque assessment than may be necessary only for detection of significant stenosis and assessment of the coronary lumen. The image characteristics of coronary plaque on CCTA images leads to higher interobserver and interscan variability for detection and quantification of plaque as compared to stenosis.\textsuperscript{116-119} The readers in both clinical and research setting should be cognizant of these limitations.

Quantitative evaluation of coronary plaque volume and characteristics is gradually becoming feasible.\textsuperscript{46, 49, 83, 111, 112, 116, 120-137} A major limitation of this approach is the time required for complete evaluation of coronary plaques in the entire coronary tree. Despite progress in automated processing, the analysis of a single dataset still can require several hours and is currently impractical for routine clinical practice. As noted above, further standardization of measurements among software platforms and standardization in reporting of quantitatively defined plaque burden are needed for broad clinical impact.

Imaging acquisition parameters and image quality have an impact on plaque quantification and characterization. This is particularly important with the current trend for use of lower peak tube potential imaging in an effort to decrease radiation dose. While this approach permits good quality luminal imaging, it has profound effects on plaque imaging and measurement of CT
attenuation of individual plaque components. The overlap of measured HU among individual noncalcified plaque components (i.e., lipid-rich versus fibro-fatty versus fibrous) is another limitation of CCTA imaging and it can be accentuated by variations in image quality, lumen enhancement, and tube potential. Moreover, for CT scans acquired at different kilovoltage (kV) settings, comparisons of plaque characteristics are problematic should be carefully considered.

**Research Areas Under-Development as to the Role of Atherosclerotic Plaque Measurement and Guided Treatment**

**Atherosclerosis-Guided Prevention – A Potential Link to Improve Patient Outcomes**

CCTA documentation of atherosclerosis provides an opportunity for the CT imager to make recommendations for preventive care. With this approach, imaging findings may lead to higher use of life saving therapies and may improve event-free survival for at-risk patients. This strategy of CCTA-guided prevention was proposed as a primary mechanism by which CCTA reduced CHD death or MI by ~40% (p=0.004) in the SCOT-HEART trial.\(^{138}\) In multiple other observational registries, evidence of initiation and intensification of preventive therapies have been reported among patients with CCTA findings of obstructive and nonobstructive CAD (Figure 8). Thus, documentation of obstructive CAD and atherosclerotic plaque findings provides an opportunity for CT imagers to make recommendations for preventive care, as described in the CAD-RADS\(^{TM}\) statement from the SCCT.\(^{96}\)

**Atherosclerotic Plaque Progression / Regression**

Both the presence as well as its extent and location of atheromatous coronary plaque predict future MACE, as is evident from a number of studies looking at plaque in its most simplistic format - CAC. However, atherosclerosis often progresses morphologically over time and is measurable using serial CCTA imaging – with progression influencing risk of hard outcomes\(^{139}\) and quantifying progression with CT Imaging might have improved prognostication and guide use of preventive therapy. A significant change in a given biomarker is understood as that which exceeds repeatability statistics or the observed changes signify worsening or improved patient
risk. Either approach is an acceptable means to define atherosclerotic disease progression. It is important to note that the evidence is limited as to our understanding of the natural history or progressive changes in atheroma across symptomatic women and men of diverse race and ethnicity; unlike the larger corpus of data that is available with CAC scanning in asymptomatic cohorts. In one report, annualized changes in total coronary plaque volume were 50.2 mm$^3$ and 21.3 mm$^3$ for diabetic and non-diabetic patients. Minimal changes were reported in noncalcified plaque volume among non-diabetics (2.5 mm$^3$ per year) as compared to more progressive disease among diabetic patients (31.2 mm$^3$). In a related report, Motoyama et al. showed that plaque progression by serial CCTA was an independent predictor of ACS. They studied 3,158 enrolled patients (mean age: 66±11 years, 70% male), the follow-up was a mean of 3.9±2.4 years with serial CCTA performed in 449 patients. Plaque progression on the second CCTA examination was an independent predictor of ACS, with adverse events occurring in 14.3% of 56 patients in the plaque progression group and 0.3% of 367 in the non-progression group (HR: 33.4 [4.1–78.0]; multivariable p=0.0006). There are few studies reporting reliable event risk thresholds for total, noncalcified, and LAP volume or progression (Table 2). However, technical protocols for CCTA (e.g., slice thickness) and the admixture of event types are quite variable and, as such, assessing progression using quantitative plaque measurement should be considered for research purposes, as this time.

**Effect of Treatments on Plaque Progression**

Evidence is available concerning the impact of lipid-lowering therapy on serial measurements of atherosclerotic plaque; albeit largely observational reports. In the recently reported in the PARADIGM (Progression of Atherosclerotic Plaque Determined by Computed Tomographic Angiography Imaging) study, 1,225 patients underwent serial CCTA at 3.8 years apart. In this study, statin-taking patients displayed a slowed rate of progression when compared with statin naïve patients. The annualized percent atheroma volume (PAV - defined as plaque volume / vessel volume) increased 1.76% for statin-taking as compared to 2.04% for statin-naïve patients.
(p=0.002) (Figure 9). A similar pattern was noted for noncalcified PAV (p<0.001). In a secondary analysis from the PARADIGM study, plaque progression was defined if plaque volume at follow-up minus plaque volume at baseline was >0.\textsuperscript{145} Participants with diabetes experienced greater plaque progression (both total and noncalcified plaque volume), particularly in adverse plaque characteristics, as compared to those without diabetes. The change in LDL was predictive of the degree and extent of atherosclerosis plaque progression in those with diabetes but not in those without diabetes. This study also suggested that tight glycemic control with lower HbA1c levels in diabetes patients was associated with a greater likelihood of plaque stabilization. In a related series, Shin \textit{et al.}\textsuperscript{146} examined 147 patients who underwent serial CCTA within a median scan interval of 3.2 years. They found that patients with LDL-C <70 mg/dl exhibited a significant attenuation in plaque progression as compared with those with follow-up LDL-C levels >70 mg/dl (12.7±38.2 mm\textsuperscript{3} vs. 44.2±73.6 mm\textsuperscript{3}, p<0.014). This, as well as other studies, demonstrate that tight LDL-C control with statins attenuates plaque volume progression.\textsuperscript{146,147} In a meta-analysis of 12 studies, intensive statin therapy reduced total plaque volume by 20.9 mm\textsuperscript{3} (or -3.6\%) compared to an increase of 15.0 mm\textsuperscript{3} was observed in controls (p=0.002).\textsuperscript{148} Statin therapy decreased noncalcified plaque volume by 7.6 mm\textsuperscript{3} and LAP volume by 5.8 mm\textsuperscript{3}. Statin users also had an increase in calcified plaque volume of 11.8 mm\textsuperscript{3}.

Additional data are also available concerning the effects of other therapies including a randomized, placebo-controlled, double-blind trial of low dose testosterone replacement in men aged ≥65 years.\textsuperscript{149} They reported that testosterone treatment was associated with an increase in noncalcified and total plaque volume. The adverse plaque progression seen among testosterone users parallels a large outcome study demonstrating increased cardiovascular (CV) events in those receiving testosterone.\textsuperscript{150} Further studies evaluating the event rates after CCTA-documented plaque progression are underway.

Despite exciting research showing the utility of imaging plaque progression current evidence is insufficient to support a clinical recommendation for serial CCTA imaging. Importantly, many
analyses focus on per patient changes whereas specific alterations within a segment or lesion may be informative for patient risk. Should a patient present with an appropriate indication for a second CCTA and a prior examination is available, imagers can document changes in the overall extent of atherosclerosis and note lesion-specific changes with a special focus on new HRP features and notable increases in noncalcified plaque volume. Importantly, although a second scan is rarely needed but if clinically indicated, similar acquisition parameters should be employed.

Future Evidence Needed in Coronary Atherosclerotic Plaque Imaging

There is extensive evidence demonstrating the feasibility of coronary atherosclerotic plaque imaging (diagnostic accuracy, detection, and measurement variability) and its role for the diagnosis of CAD and assessment of future CAD risk. Future studies should include the areas, which require a more robust quality of evidence and data that are more comprehensive and validated extensively. There is a need for data demonstrating standardization and high reproducibility of coronary atherosclerosis evaluation. Creation of standards (e.g. RSNA QIBA - Quantitative Imaging Biomarkers Alliance) for the coronary atherosclerotic plaque assessment will be important.\textsuperscript{151} In the future, vendors in the area of plaque assessment software will need to demonstrate adherence to these standards. There is an additional need for improved workflow in quantitative plaque assessment and continued decrease in the time needed for basic and advanced plaque assessment. Future studies will have to demonstrate if artificial intelligence techniques can improve performance of CCTA for coronary atherosclerotic plaque assessment. Ultimately, the accreditation and quality assurance programs for cardiac CT laboratories should include at least qualitative and potentially quantitative evaluation of atherosclerotic plaque assessment in addition to stenosis and other areas (e.g., structural heart interventions, perfusion, etc.).

Several areas of atherosclerotic plaque imaging will require further investigation in order to strengthen their role in clinical practice. Future studies should demonstrate changes in clinical
management (e.g. increased use of preventative therapies, decrease in downstream testing) and improvement of clinical outcomes (decrease in CAD events when patients are managed based on coronary plaque evaluation). Investigation is required into the interaction and complementary role of coronary plaque assessment with functional assessment of coronary lesions (e.g., FFR-CT), not only with ischemia by FFR or FFR-CT but also microvascular dysfunction or vasospasm in ischemia with nonobstructive coronary arteries.\textsuperscript{152, 153} There is also increasing interest in the measurement of perivascular adipose tissue (PVAT), measured in the range of attenuation indices from -190 to -30 HU.\textsuperscript{154} PVAT has been shown as a surrogate of inflammation and is additive in terms of risk assessment over and above CAD stenosis severity.\textsuperscript{155, 156} The methods for measurement have evolved and additional standardization of volumetric assessment is necessary. Moreover, additional clinical evidence concerning the interactions between PVAT and atherosclerotic plaque would further support routine clinical measurement.

**Guideline-Concluding Statements**

There is ever-expanding evidence as to the role of imaging of atherosclerotic plaque and the importance of broadening the scope of CCTA to more than detecting obstructive lesions. This clinical guideline provides a synopsis of available evidence and targets essential elements to include in a structured CCTA report. Critical elements needed for improved structured reporting include documentation of the presence of atherosclerotic plaque (descriptive statements, CAD-RADS\textsuperscript{TM} categories), the SIS, HRP features (e.g., LAP, PR), and the CAC score (when performed). Imaging of coronary atherosclerosis is an area of active research and we anticipate that this statement will need to be updated to include guidance on improved quantitative approaches for CCTA plaque measurement. The documentation of atherosclerosis on CCTA provides an important opportunity to make recommendations for preventive care and to initiate and guide an effective care strategy for at-risk patients.
References


122. de Graaf MA, Broersen A, Ahmed W, Kitslaar PH, Dijkstra J, Kroft LJ, Delgado V, Bax JJ, Reiber JHC and Schulte AJ. Feasibility of an automated quantitative computed tomography


Table 1. Evidentiary Standards for Clinical Application of CCTA Measures of Atherosclerotic Plaque

<table>
<thead>
<tr>
<th>Quality Evidence Required for CCTA Atherosclerotic Plaque:</th>
<th>Quality Standard Met:</th>
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<tbody>
<tr>
<td>o The Evidence Is Valid and Highly Correlated with Invasive or Pathologic Measures</td>
<td>□</td>
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<tr>
<td>o Atherosclerotic Plaque Can Be Reliably Measured Across Patient Subgroups (Including by Age, Sex, Body Habitus) and by Physicians of Variable Expertise and Software Platforms</td>
<td>X</td>
</tr>
<tr>
<td>o The Observational Findings Reveal a High Accuracy for Detection of At-Risk Patients; Including Validation Reports Across a Range of Patient Subgroups and Settings (e.g., Emergency Department or Outpatient Cohorts)</td>
<td>□</td>
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<tr>
<td>o Atherosclerotic Plaque Has Established Prognostic Significance with Defined Risk-Based Thresholds</td>
<td>+/-</td>
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<tr>
<td>o Targeted Preventive Strategies with Definable Clinical Outcomes, Including Surrogate Outcome Data (i.e., serial CCTA for identifying atherosclerosis progression), Across Diverse Preventive Regimens and In a Secondary Prevention Strategy for Symptomatic Patients</td>
<td>X</td>
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</table>
**Table 2.** Quantitative Measurement of Atherosclerotic Plaque – Risk-Based Thresholds Reported in Patients Experiencing CAD Events

<table>
<thead>
<tr>
<th>Author</th>
<th>N=</th>
<th>Follow-up</th>
<th>Event Definition</th>
<th>n=</th>
<th>Total</th>
<th>LAP</th>
<th>Noncalcified Plaque</th>
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<tbody>
<tr>
<td>Deseive(^{142})</td>
<td>432</td>
<td>5.6 years</td>
<td>Death, ACS, or Late Revascularization</td>
<td>44</td>
<td>110.5</td>
<td></td>
<td></td>
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<tr>
<td>Deseive(^{141})</td>
<td>1,577</td>
<td>5.5 years</td>
<td>Death or ACS</td>
<td>30</td>
<td>2.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hell(^{60})</td>
<td>2,748</td>
<td>5.2 years</td>
<td>CAD Death</td>
<td>32</td>
<td>10.6</td>
<td>146.0</td>
<td></td>
</tr>
<tr>
<td>Dwivedi(^{143})</td>
<td>72</td>
<td>~4.0 years</td>
<td>Death or MI</td>
<td>36</td>
<td>4.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tesche(^{59})</td>
<td>92</td>
<td>1.0 year</td>
<td>CAD Death, MI, UA leading to Revascularization</td>
<td>46</td>
<td>122.6</td>
<td></td>
<td>67.3</td>
</tr>
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**N=6,089**

Average = 4.5 years

n=234 (0.9% / year)

Average = 116.6

Average = 5.8

Average = 106.7

Abbreviations: ACS: Acute Coronary Syndrome; CAD: Coronary Artery Disease; MI: Myocardial Infarction; UA: Unstable Angina.
**Table 3. SCCT Summary Recommendations for Visualization and Measurement of Coronary Atherosclerotic Plaque**

<table>
<thead>
<tr>
<th>Recommendation</th>
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<tr>
<td>The interpreting physician should include integrate evidence of coronary artherosclerotic plaque into the laboratory standard report.</td>
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<tr>
<td>For patients with evidence of coronary atherosclerotic plaque, it is recommended that the physician report the presence of HRP (when present) in order to improve risk stratification and guide clinical management decisions.</td>
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<td>For patients with evidence of coronary atherosclerotic plaque, it is recommended to use the CAD-RADS modifier “V” if a coronary plaque demonstrates 2 or more HRP features in the CCTA final interpretation, or to describe what HRP characteristics are present.</td>
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<td>For patients with evidence of coronary atherosclerotic plaque, the CT imager should consider including the SIS in the CCTA final interpretation.</td>
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<td>In patients undergoing a non-contrast scan for calcium quantification as part of their CCTA examination, it is recommended to report the total CAC score and the associated risk category of 0, 1-99, 100-299, and ≥300, respectively.</td>
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<td>For patients with evidence of coronary atherosclerotic plaque, the conclusion of the report should include a statement regarding the overall amount or extent of atherosclerotic plaque. This can be based on a visual assessment (acknowledging the aforementioned limitations); the CAC score (if performed); or a semi-quantitative assessment of the number of coronary segments with plaque using the SIS.</td>
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<tr>
<td>Royalties: Elsevier; Grants and research support: NIH; AHA, NIH; General Electric; Stock and stock options and employment: Cleerly Inc.; Consultant/Honoraria: HeartFlow; Software royalties from Cedars-Sinai Medical Center</td>
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Central Illustration: SCCT Stages of Atherosclerosis Detected by CCTA
Figure 1. Meta-Analysis of the Prevalence and Clinical Outcomes of Patients Undergoing CCTA with Nonobstructive CAD.
Cardiac events were evaluated for nonobstructive CAD (1-49% Stenosis) among patients presenting with suspected disease in 17 published reports (N=49,957) with a median of 2.5 years of follow-up. There is an admixture of event types (all-cause or CAD mortality, ACS, or Revascularization) and length of follow-up (Range: 1.7-10.0 years). There is an 8-fold higher rate of events among patients with nonobstructive CAD as compared to those with No Stenosis or Plaque. Note that the two figures have different y-axis ranges.
Figure 2: Stable and high-risk plaque pathology as characterized by intravascular ultrasound (IVUS) and optical coherence tomography (OCT). Stable plaque (A-D) or fibroatheroma is characterized a variable lipid core covered by a thick fibrous cap. High-risk plaque (E-J) or thin-cap fibroatheroma usually shows a large total plaque volume, necrotic core volume, positive remodeling and a thin fibrous cap; these plaques are usually substantially inflamed. IVUS identifies plaque volume, necrotic core and positive remodeling while OCT is best suited to define thin fibrous caps. Modified from Otsuka et al. Nature Reviews Cardiology 2014.
Figure 3: Hierarchical Importance of Morphological Characteristics of Plaque Vulnerability by Recursive Partitioning Analyses. Pathological characterization of nearly 300 stable (green), high-risk (blue) and disrupted (red) plaques revealed fibrous cap thickness (FCT) to be the most important determinant of plaque vulnerability (left); FCT was <85mm in high-risk and <55mm in disrupted plaques. Since FCT can only be assessed by an invasive procedure, reanalysis without including FCT suggested the extent of plaque inflammation and the magnitude of the necrotic core to be the best determinants (right). Both these characteristics can are amenable to CT angiography though PET imaging is more informative about vascular inflammation. Modified from Narula et al. JACC 2013.
Figure 4: CCTA characterization of plaque composition. CCTA characteristics of stable (A), ruptured (B) and high-risk (C) plaques. Stable plaque in a patient with stable angina has no positive remodeling or low-attenuation plaque (LAP); lumen is critically stenotic (green arrow). In a patient with NSTEMI, the plaque shows LAP (red) and positive remodeling (yellow). Such high-risk plaques have been called as 2-feature positive plaques. In the CT angiogram below, a patient in ER presented with the 2-feature positive plaque but was ruled out by serial ECG and troponin. The high-risk plaque is just proximal to S1 branch. The patient reported to ER again 6 months later with STEMI and the culprit lesion was indeed proximal to S1. Modified from Motoyama et al. JACC 2007 and 2009.
Figure 5: CT angiography-verified plaque composition and clinical outcomes. CT angiography-based 2-feature positive plaques demonstrate a 45-fold higher likelihood of cardiac events as compared to 2-feature negative plaques at 2 years of follow-up (A) and 9-fold higher event rate up to 10 years of follow-up (B). The extent of necrotic core as represented by LAP volume >4% (C), added severity of luminal stenosis (D) and plaque progression in serial CT angiography (E) improve the discriminatory value of CT angiography. Modified from Williams et al. Circulation 2020 and Motoyama et al. JACC 2015.
Figure 6. Quantitation of Atherosclerotic Plaque. (A) The analysis includes 3D measurement of all major epicardial vessels and branches (>1.5 mm); measurements are derived from 0.5 mm cross-sectional slices for characterization of atherosclerotic plaque and lumen. (B) Current evidence reveals that quantification of atherosclerotic plaque using CCTA is highly concordant with IVUS measurements as demonstrated by meta-analysis of 42 studies including 1360 patients; difference between CCTA and IVUS plaque volume was 1.5 mm³ (p=NS), and AUC for CCTA vs. IVUS plaque volume was 0.97. Modified from Fischer et al. JCCT 2013.
Figure 7. Nomograms for use of the segment involvement score (SIS) Across Diverse Ages. Age percentiles are based on SIS derived from 11,418 men (A) and 9,714 women (B).
Figure 8. Summary of Observational Evidence as to the Absolute Change in Statin or Aspirin Prescription. According to CCTA Results. (A) The average change with statin prescription was +0.2%, +22.2%, and +28.6%, and (B) +7.5%, +25.2%, and +38.2% for aspirin prescription, for no plaque, nonobstructive and obstructive CAD, respectively.
Figure 9. Effects of Statins on Coronary Atherosclerotic Plaque. In the Progression of Atherosclerotic Plaque Determined by Computed Tomographic Angiography Imaging [PARADIGM] study, paired CT scans performed 2 years apart were available in 1,255 patients (A). In statin-taking patients, while there was a 23% increase in calcified plaque volume, the non-calcified plaque volume decreased by 54%. In addition to the changes in overall, calcified, and non-calcified plaque volume, there was an observed 35% decrease in incident HRP. Modified from Lee et al. JACC CV Imag 2018.