10. 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 require a thickness of 0.5–1 mm to be detected by CCTA.[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.[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.[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 and should be carefully considered.

11. Research areas under-development as to the role of atherosclerotic plaque measurement and guided treatment

11.1. 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 haves been reported among patients with CCTA findings of obstructive and nonobstructive CAD (Fig. 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™ statement from the SCCT.[96]

11.2. 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[139] 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.[140,157] 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). Otaki et. al. reported changes in quantitative coronary plaque measurements in a study of 151 patients undergoing serial CCTA for clinical purposes (interval 4+/- 2 years), comparing patients with and without a decrease in LDL >10% from baseline. In the decreased LDL group there was regression in all components of noncalcified plaque on the follow-up study, whereas, in the no LDL decrease group, progression of all of the noncalcified plaque components was observed (< 0.001 for all). In a related report, Motoyama et al.[38] 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 3
SCCT summary recommendations for visualization and measurement of coronary atherosclerotic plaque.

Recommendation:
The interpreting physician should include integrate evidence of coronary artherosclerotic plaque into the laboratory standard report
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.
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.
For patients with evidence of coronary atherosclerotic plaque, the CT imager should consider including the SIS in the CCTA final interpretation.
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.
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.