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Coronary Artery Calcium Testing—Too Early, Too Late, Too Often

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IMPORTANCE Traditional risk factors, enhancing factors, and risk scores help clinicians assess atherosclerotic cardiovascular disease (ASCVD) risk for primary prevention. The latest cholesterol guidelines suggest measuring coronary artery calcium (CAC) score by computed tomography (CT) in those at intermediate risk when there is uncertainty about statin initiation for primary prevention. CAC testing can improve both risk estimation and adherence to cardiovascular risk–reducing behaviors.

OBSERVATIONS As measuring CAC score has become more widely available, this article focuses on 3 situations where CAC testing may be omitted or deferred until a time when CAC testing can provide clinically useful information. Three clinical scenarios to facilitate the clinician-patient risk discussion are as follows: (1) when CAC testing is too early, (2) when CAC testing is too late, and (3) when CAC testing is repeated too often. The timing of CAC testing sits within the decision point of lipid-lowering therapy use. High-risk young adults may face an elevated lifetime risk of cardiovascular disease despite a CAC level of 0, whereas older adults may not see an expected benefit over a short time horizon or may already be taking lipid-lowering therapy, rendering a CAC score less valuable. Integrating a CAC score into the decision to initiate lipid-lowering therapy requires understanding of a patient's risk factors, including age, as well as the natural history of atherosclerosis and related events.

CONCLUSIONS AND RELEVANCE These clinical scenarios reflect when consideration of CAC score is of use and when it is not. Although CAC testing is becoming more widely available and sought after by clinicians and patients alike, it is only as useful as the clinical context. Understanding when assessing CAC score is too early to effectively rule out risk, too late to influence decisions, or too often to yield clinically relevant information provides important insights that optimize the clinical utility of this potentially valuable prognostic tool.

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Atherosclerosis is a dynamic disease process that often starts as fatty streaks and intimal thickening and subsequently progresses to fibroatheroma formation through multiple steps. Calcium deposition is radiographically apparent by conventional imaging techniques late in the disease process and thus reflects advanced atherosclerosis and aggregate plaque burden (calcified and uncalcified). Measuring the calcium level within atherosclerotic lesions can improve individual-level risk prediction for future atherosclerotic cardiovascular disease (ASCVD) among adults. Coronary artery calcium (CAC) score can be quantified in less than 15 minutes by a computed tomography (CT) scan with low-level (approximately 1 mSv) radiation exposure. An individual's CAC score is generated from the Agatston score, which is the summation of each CAC lesion multiplied by an attenuation factor.[1] This absolute score is then compared with the normative distribution by age, sex, and race—largely derived from the Multi-Ethnic Study of Atherosclerosis (MESA) study—to express the burden of CAC in terms of age, sex, and race-specific percentiles.[2]

Multiple studies have documented the association between traditional ASCVD risk factors (eg, low-density lipoprotein cholesterol [LDL-C], diabetes, smoking) and static CAC measurements, as well as CAC progression.[3] Beyond this, an individual's CAC score can offer added insight into incident ASCVD risk—particularly among adults without a history of ASCVD and not taking statin therapy. As adults with a low burden of traditional ASCVD risk factors represent a large population, they contribute numerous ASCVD events. Individuals with a low-risk profile by traditional risk factors may still have CAC.[4,5] Among these individuals, CAC portends increased risk of an event despite few traditional risk factors and improves discrimination of risk.[6,7] Knowing an individual's CAC score may potentially help with reclassifying the risk predicted by the pooled cohort equations (PCEs), even among those with a low burden of traditional risk factors.[8,9] Although CAC can help reclassify ASCVD risk in some low-risk patients, evidence is limited regarding whether this reclassification translates into significant reductions in clinical events. For example, in the study by Akintoye et al,[9] only 5.7% of low-risk patients were reclassified. Although these individuals had an 8.7% higher event rates, it is unclear if this net reclassification and event reduction would justify widespread screening given the potential costs.[9]

However, not all investigators agree that a CAC score can provide clinical benefit. In part, this is due to limited randomized clinical trial (RCT) data studying the impact of CAC score on clinical outcomes. Intermediary outcomes, however, such as LDL-C levels and statin initiation, have demonstrated improvement with CAC screen-

ing. In the Early Identification of Subclinical Atherosclerosis by Noninvasive Imaging Research (EISNER) study of 2137 adults randomized to CAC screening and the Coronary Artery Calcium Score: Use to Guide Management of Hereditary Coronary Artery Disease (CAUGHT-CAD) study of 450 adults randomized to notification of CAC score, LDL-C levels were lower in the intervention arm of both studies.[10,11] Of note, in the EISNER study, among adults with a CAC score greater than or equal to 400, follow-up care resulted in a higher rate of all forms of noninvasive stress testing, as well as angiography and revascularization resulting in significantly greater health care costs, particularly for procedures.[10] Investigators in the Incidental Coronary Calcification Quality Improvement Project (NOTIFY-1) study found that statin initiation was 44.3% higher among adults randomized to notification of incidental CAC compared with those who were not.[12] Additionally, there was a higher rate of downstream testing in the NOTIFY-1 study of 15.1% compared with 2.3.% among adults notified of CAC score, largely driven by noninvasive stress testing (11.6% in the notified arm vs 2.5% usual-care arm). The potential for a CAC measurement to prompt further testing is a concern, particularly in a clinical scenario where it was not indicated as outlined subsequently, as well as among adults with high baseline CAC score among asymptomatic adults where inertia may drive further interventions. However, given the limited sample size of these studies and available data, if and how this translates into clinical event rate reduction remain unknown. Further research leveraging clinical variability in real-world practice patterns with associated ASCVD compared with unnecessary interventions outcomes may help complement RCT data to inform practicing clinicians on the utility vs possible negatives of obtaining a CAC measurement.[12] To date, the Danish Cardiovascular Screening (DANCANVAS) trial is the largest trial attempting to answer whether CAC screening reduces cardiovascular events. However, this remains inconclusive, as all-cause death was not reduced, although events were lower among adults aged 65 to 69 years and for stroke outcomes.[13] However, an increasing myriad of data supports that adding a CAC score to traditional cardiovascular risk factors can improve predictive modeling of future ASCVD event risk in the appropriate clinical context.[3,14] Indeed, context is very important. Among adults with a PCE 10-year calculated ASCVD risk of 5% to 7.5% and 7.5% to 20%, a CAC score of 0 correlated with an actual event rate of 1.5% and 4.5%, respectively—substantially lower than that calculated by the PCE alone.[3,15] For specific adults with a CAC score of 0, their risk can be better inferred and reclassified to a lower risk category.[16] In contrast, for similar adults without a history of ASCVD and a 10-year calculated ASCVD risk of 7.5% to less than 20% (ie, intermediate risk), any measured CAC portends an increased likelihood of disease. Indeed, a score of 100 or higher or those in the upper 75th percentile should promote strong consideration of statin initiation. Determining percentile of risk may be especially important in younger patients (eg, <50 years of age), where the presence of any CAC represents accelerated atherosclerosis for this age group. How CAC is scored and how that score translates into clinical action are critical to determining when a CT CAC score is warranted. The 2018 American Heart Association (AHA)-American College of Cardiology (ACC)-Multisociety (MS) guidelines recommend that when a risk decision is uncertain, a CAC score can improve specificity of primary prevention risk assessment in adults 40 to 75 years of age without diabetes or an LDL-C level greater than or equal to 190 mg/dL (to convert to millimoles per liter, multiply by 0.0259), at an intermediate and sometimes borderline 10-year calculated ASCVD risk.[16] Additionally, the guidelines provide clinical examples for when a CAC score should be measured and when a score of 0 can help guide care (**Table 1**).[16]

Table 1. Clinical Scenarios[a] for When a Coronary Artery Calcium (CAC) Level of 0 May Impact Clinical Decision-Making

Scenario	Description
1	Statin-naive patients hesitant to initiate statin therapy and require more information regarding their individual risk vs benefit of statin therapy
2	Patients previously trialed on statin therapy who were unable to tolerate statin therapy due to side effects and are considering retrialing statin therapy
3	Adults aged 55-80 y for men or 60-80 y for women who do not have significant cardiovascular risk factors and are unclear whether a statin is likely to benefit them
4	Adults aged 40-55 y with a calculated 10-y risk of ASCVD of 5% to <7.5% by the PCE that have other factors that increase their ASCVD risk

Abbreviations: ASCVD, atherosclerotic cardiovascular disease; PCE, pooled cohort equation.

[a] Clinical scenarios adapted from the 2018 Guideline on the Management of Blood Cholesterol: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines.[16]

As with all diagnostic tests, a clinician must determine if they are applying the right tests to the right patients at the right time. As measuring CAC score has become more widely available, we are concerned with specific situations where CAC testing may not provide clinically useful information and may impose potentially significant emotional, physical, and financial harm among asymptomatic adults through increased downstream testing by means of unnecessary stress testing, coronary catheterization, and revascularization despite a lack of symptoms or clinical indication.[17-19] Herein, we review the evidence for an important consideration: when is a CAC score measurement likely to be obtained too early, too late, or even, too often (**Table 2**)?[20]

Measuring CAC Score Too Early

Young age is an important consideration in determining when CAC testing is premature, but there are other considerations for whom a CAC score does not provide actionable information. The 2018 AHA-ACC-MS guidelines and 2017 Society of Cardiovascular Computed Tomography expert consensus do not consider measuring CAC in primary prevention adults younger than 40 years.[16,21] These recommendations were further carried over into the 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease.[22]

Although these guidelines highlighted the role of a CAC score of 0, clinicians should be aware of those patients where obtaining a CAC score of 0 is unlikely to provide useful information. To determine eligibility for CAC testing in a young adult requires a thought process that considers the yield and the likelihood of an actionable result. In major cohort studies including the MESA trial, Heinz Nixdorf Recall study, Rotterdam Study, Framingham Heart Study, the Coronary Artery Risk Development in Young Adults (CARDIA) study, Jackson Heart Study, and the Women's Health Initiative, the majority of CAC scores were obtained among middle-aged adults. Although CAC

Table 2. When Coronary Artery Calcium (CAC) Testing May Be Too Early, Too Late, or Too Often in 2024[a]

Variable	Too early	Too late[b]	Too often[c]
Clinical scenarios	• Males aged <40 y without risk factors • Females aged <50 y without risk factors • Males with DM age <35 y • Females with DM age <45 y • Below 5% ASCVD risk unless a strong lifetime risk burden such as family history of premature coronary disease or current smokers	• Already on statin therapy • CAC does not measure statin efficacy against reducing plaque volume • Age >65 y with many ASCVD risk factors in whom a treatment decision is not uncertain • Age ≥80 y	• Repeating CAC score if CAC ≥300 for progression of disease • Repeating CAC score if CAC ≥100 and on risk-reduction therapy • Repeating a CAC score in older patients >75 y if CAC = 0[20]

Abbreviations: ASCVD, atherosclerotic cardiovascular disease; DM, diabetes mellitus.

[a] These suggestions cannot take the place of an individualized clinical assessment of a patient and their risk status. They are intended to aid the clinician in their discussion of whether the CAC score test if obtained is likely to provide information that will change therapy.

[b] Too late to change clinical management.

[c] There may be individual circumstances where a repeat CAC score is warranted, but this requires careful consideration and should not be performed routinely.

Figure. Schematic of the Expected Change in Atheroma Composition and Coronary Artery Calcification With Statin Use

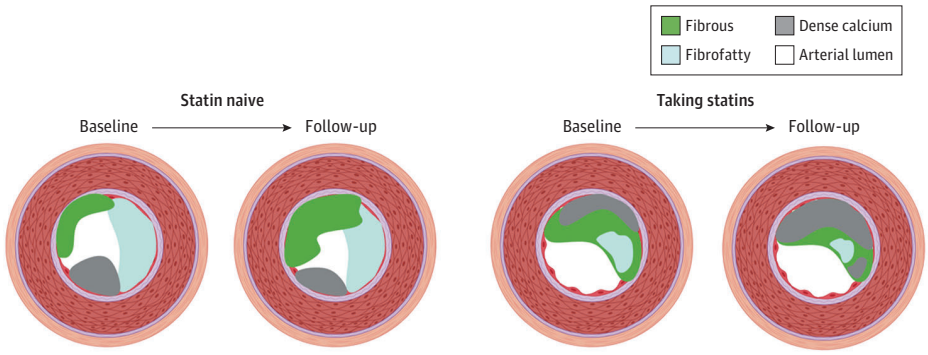

Figure adapted.[32]

was measured as young as 32 years in the CARDIA study, in most cohorts, CAC was not measured until at least 45 years of age.[3,23-26] In the CARDIA study, the presence of CAC predicted fatal and nonfatal CAD after 12.5 years of follow-up, but only 1 in 10 participants in the age range of 32 to 46 years had a CAC score greater than 0.[27] Data from the CAC Consortium suggest that the optimal age for an initial CAC score may be 42.3 and 57.6 years in men and women, respectively, without significant risk factors, and even younger for men and women with diabetes, 36.8 years and 50.3 years, respectively.[28] However, given that diabetes confers a significantly elevated ASCVD risk, especially in younger patients under 40 years, a CAC score of 0 in a young person with diabetes may in fact be falsely reassuring as those with a CAC score of 0 have a similar risk of major adverse cardiac events as those with a score of greater than 0 to 100.[29] A CAC score of 0 in a high-risk young adult with diabetes with long durations of type 2 (≥10 years) or type 1 (≥20 years), microangiopathic complications, or a positive ankle brachial index should not delay appropriate statin use.[30] Indeed, a CAC score of 0 may offer a chance of a high-risk patient to influence subsequent development of atherosclerosis and thus represent an opportunity to keep their risk low through the prevention of atherosclerosis.[31]

In general, a CAC score in an individual younger than 40 years is not likely to provide actionable data. Although knowledge of a CAC score may improve adherence to guideline recommendations if CAC score is greater than 0, clinicians should note that that in the presence of cigarette smoking, genetic hypercholesterolemia, or high risk factor burden, a CAC score of 0 in young adults does not indicate that statin therapy can be automatically deferred.[16] Among young adults with risk factors for ASCVD, addressing the individual causal determinants of disease before consideration of noninvasive imaging given insufficient data and longer time horizon by which CAC develops is important.

Measuring CAC Score Too Late to Be Helpful

In some clinical circumstances, a CAC score loses its ability to meaningfully inform clinical decision-making regarding statin assignment—such as when an older adult's expected lifespan precludes time for benefit or when risk is already high enough that a CAC measurement is unlikely to change therapy. For example, based on data from the MESA study, among adults with high baseline risk, eg, a 10-year risk score of 20% or greater, a CAC score of 0 reduces the estimated risk to approximately 10%, but this is still an estimated risk level where a patient derives benefit from statin therapy.[15] Also, patients with elevated CAC scores while taking statin therapy are unlikely to benefit because it is unclear if the increase in CAC scoring is due to increased density of the calcification or worsening of the plaque, and the decision to start statin therapy has already been made, as outlined in the **Figure**.[32] Beyond a lack of clinical utility for these patients, this may create unwarranted patient anxiety in our experience. Certainly, above age 80 years, there is little guidance for measuring CAC level at present, which is much more common among older adults. In a cohort of 614 adults older than 65 years (mean age, 80 years), more than 90% had a CAC score greater than 0 with an overall mean of 671 Agatston units.[33] As such, routine measure-

ment of CAC score in adults 80 years or older is unlikely to inform clinical decision-making regarding evidence-based statin use.

Another scenario by which CAC measurement comes too late is among adults for whom the decision to take a statin has already been made. A CAC score is best used to inform the decision of statin initiation, not titration. Anecdotally, we have seen patients where a CAC score was ordered yearly in those taking statin therapy presumably to determine if more aggressive therapy is needed. Importantly, the expected increase in CAC level among patients taking statin therapy may actually function as negative feedback if not carefully explained to the patient. Statin therapy has consistently been associated with an increase in CAC level. The increase in CAC level is a result of the composition of the atheroma changing to a greater relative area of stable calcified plaque (Figure).[32] A pooled analysis[34] of 8 clinical trials assessed the effects of statin therapy on atheroma size and composition by intravascular ultrasound between 18 and 24 months after statin initiation and a baseline CAC level was measured. In this study, high-intensity statin therapy reduced atheroma size by approximately 1.5% and led to a greater overall increase in calcification compared with participants who received a placebo medication.[34] Participants taking low-intensity statin therapy also demonstrated greater calcification compared with placebo. The change in calcification appears to be dose dependent. Thus, high-intensity statin therapy exhibits a greater overall increase in CAC level when compared with the increase in CAC seen with low-intensity statin therapy.[34] The Progression of Atherosclerotic Plaque Determined by Computed Tomographic Angiography Imaging (PARADIGM) study compared adults who were taking statin therapy with those who were statin naive. The mean (SD) annual progression of calcified atheroma volume was 1.27 (1.54) mm^3 vs 0.98 (1.27) mm^3 per year, respectively ($P < .001$).[32] Calcification of atheroma is an anticipated effect of statin therapy and a result of positive remodeling.[35] Given the overwhelming evidence of the impact of statin therapy on reducing atherosclerosis plaque volume and associated improved clinical outcomes despite its effect on increasing calcification, there appears to be little value in measuring CAC score after statin therapy is initiated.

Measuring CAC Score Too Often

Repeat imaging must be paired with consideration of how this would change management. The Society of Cardiovascular Computed Tomography expert consensus recommends that repeat imaging may be beneficial among adults with a CAC score of 0 or CAC score greater than 0 and less than 100 in 5 years or 3 to 5 years, respectively.[21] The 2018 AHA-ACC-MS guidelines provide a slightly longer range of 5 to 10 years between repeat imaging as a reasonable interval before measuring another CAC score if the initial CAC score was 0 and the patient has no major risk factors (ie, smoking or incident diabetes).[16] This evidence is largely based on longitudinal cohort studies, as RCT evidence is lacking. For example, neither the Heinz Nixdorf Recall study nor the Cooper Center Longitudinal study found significant clinical improvement in risk prediction based on CAC progression.[36,37] However, a repeat CAC score of 0 at 5 years is associated with a very low risk of cardiac and cardiovascular events.[36] Data from the MESA study supported the findings of the Heinz Nixdorf Recall study and Cooper Center Longitudinal study. Among adults with a CAC score less than 100 in the MESA study, the cumulative incidence of ASCVD was less than 7.5% across all races and ethnicities, as well as adults younger than 75 years over 10 years.[38] Among CAC Consortium participants with a CAC score less than 100, the absolute annual mortality rate was less than 1.0 per 1000 suggestive of a very low event rate.[39] Data from these longitudinal cohorts are the basis by which a 5-year interval has been suggested for interval screening when indicated.

An important example is the adult with an initial CAC level greater than or equal to 75% percentile for age, sex, and race and ethnicity. We are not aware of data showing benefit to the patient by knowing that their CAC has increased. Indeed, there does not appear to be a threshold of CAC where the trajectory of ASCVD risk flattens. Cardiovascular disease risk among CAC Consortium participants with a CAC score greater than or equal to 1000 was 5.04-fold greater than those with a CAC score of 0 and 1.71-fold greater than those with a CAC score of 400 to less than 1000.[40] The magnitude of increased risk among these categories of CAC scores (0, 400 to <1000, and ≥1000) were similar among MESA study participants.[41] CAC data suggest that LDL-C–lowering therapy in those with a CAC score greater than or equal to 300 should be prescribed with comparable LDL-C goals as adults with a history of ASCVD. A repeated CAC score in these adults, especially for those taking statin therapy, does not provide an accurate estimate of efficacy and would not provide a rationale for a change in therapeutic intensity. Adults with a CAC score of 100 or greater have a substantially elevated risk of future ASCVD and are unlikely to gain benefit from deferring intensive risk factor reduction with statin and possibly aspirin therapy.[42-45] When a repeat CAC score is obtained, there should be a clinical decision under consideration.

To exemplify the concepts of when CAC testing is too early, too late, or too often, we present 3 clinical vignettes from our clinical practice. We hope that these examples provide clear clinical guidance with regard to the discussion of when CAC testing is appropriate.

Clinical Vignettes of CAC Testing

Patient A

A 38-year-old woman who is gravida 2, para 2 with an LDL-C level of 150 mg/dL presents to the clinic today to discuss whether she should obtain a CAC test.

The patient has no significant medical history, is not taking an oral contraceptive medication, and has never smoked cigarettes. Her recent lipid panel revealed a total cholesterol level of 235 mg/dL (to convert to millimoles per liter, multiply by 0.0259), triglyceride level of 200 mg/dL (to convert to millimoles per liter, multiply by 0.0113), high-density lipoprotein cholesterol (HDL-C) level of 46 mg/dL (to convert to millimoles per liter, multiply by 0.0259), LDL-C level of 150 mg/dL, and a non–HDL-C level of 190 mg/dL. A lipoprotein(a) level was measured and was 125 nmol/L (to convert to milligrams per deciliter, multiply by approximately 0.4). Her hemoglobin A_{1c} level was 5.3%. She notes that one of her parents had a heart attack in their 50s. She is considering taking a statin but is wondering if she should obtain a CAC score first. However, given her age, the likelihood of a CAC score greater than 0 was low, and despite her risk factors, testing for CAC at age 38 years was likely to be too early to be useful, particularly as the patient's stated goal was to minimize fu-

ture risk.[27] We reviewed her family history of premature ASCVD, dyslipidemia, and elevated lipoprotein(a) level and discussed that our traditional approach to estimating risk through use of the PCE with consideration of CAC measurement is not validated at such a young age. After shared decision-making, a moderate-intensity statin was started for ASCVD risk prevention given the patient's goal of minimizing the risk of ASCVD into her middle and later years.

Patient B

An asymptomatic middle-aged man with hypertension with a predicted 10-year risk of ASCVD of 22%, leading to obtaining a CAC measurement, which was 492, with a comment that this was in the 98th percentile for his age, sex, and race, and for further testing as indicated. These results prompted significant patient distress, and he subsequently had positron emission tomography stress testing, which demonstrated a small inferoseptal area of ischemia with preserved myocardial blood flow reserves. He underwent angiography and received a drug-eluting stent to the distal right coronary artery. The patient never experienced symptoms. The patient remained concerned about his risk of an ASCVD event and self-referred.

Before CAC testing, the patient was already considered to be high risk by the PCE, irrespective of his CAC measurement, although he remained asymptomatic. His off-treatment lipid panel was notable for an LDL-C level of 110 mg/dL and an apolipoprotein B level of 132 mg/dL (to convert to milligrams per liter, multiply by 0.1). Both responded to rosuvastatin, 20 mg, and ezetimibe, 10 mg, with follow-up testing showing an LDL-C level of 51 mg/dL and an apolipoprotein B of 63 mg/dL.

In this case, obtaining a formal CAC measurement comes too late to provide any information useful in determining the patient's care. The patient is at a high risk of future ASCVD events and, even had his CAC been 0, his anticipated ASCVD risk would exceed 10% and warrant statin therapy. Furthermore, the CAC measurement prompted unnecessary downstream testing and procedures with potential for physical harm and coming at a financial and emotional cost to the patient. Although a CAC score may have been potentially useful in someone hesitant to begin taking a statin, in this statin-naive patient, the pretest positivity was too high to warrant CAC measurement and led to unnecessary testing, which was not indicated given that he was asymptomatic.

Patient C

A 51-year-old man presents to your clinic with a history of hypertension and a recent diagnosis of diabetes with albuminuria and evidence of early retinopathy. When he was last seen in clinic 2 years prior, a CAC test had been performed, at which time no CAC was seen. The patient was curious about performing another CAC test.

When seen previously in clinic, the patient had been resistant to statin therapy, despite an LDL-C level of 154 mg/dL. However, with the diagnosis of diabetes complicated by kidney dysfunction and retinopathy, he now warrants statin initiation. The patient initiates treatment with rosuvastatin, 40 mg, and achieves a 60% reduction in LDL-C level. In this scenario, there is no benefit to repeating his CAC measurement. First, the 2-year time horizon is likely not long enough for there to be a significant increase in CAC score. More importantly, this patient now has specific indication of statin therapy, and a repeat CAC score would do little to guide management. Had this patient not developed diabetes, it would have been reasonable to repeat a CAC measurement 3 to 5 years after his initial scan.

Conclusions

Measurement of CAC score among adults without ASCVD and of intermediate risk, as well as borderline risk in certain cases, can help refine risk stratification when a clinical decision is uncertain. CAC testing can also be useful in other selected patient populations where a CAC score of 0 may guide clinical management (Table 2). However, measuring CAC too early may prove a myriad of issues. As CAC often develops later in adulthood, young adults with significant risk factor burden may be falsely reassured about their long-term risk with a CAC of 0, despite the time horizon of CAC development, lack of validation in younger cohorts, and higher long-term risk. False reassurance provided by premature testing for CAC may diminish efforts to promote important lifestyle-based cardiovascular risk reducing interventions. In contrast, measuring CAC too late—whether in an older adult over 80 years or in those already on statin therapy – may prove to be an exercise in futility, generating data that adds little to clinically relevant care. Although there are patients such as those with a CAC score of 0 who benefit from repeat measurement in 3 to 5 years to help guide specific clinical decision making, those with a CAC score greater than or equal to 100 and/or a CAC score in the 75th percentile or greater should proceed to risk reduction treatment with statin therapy. CAC represents overt atherosclerotic disease and conveys more than the probability of disease unlike a calculated risk score. After statin initiation, repeated CAC imaging is not warranted to guide titration of statin therapy. Clinicians should explain to patients why a CAC score in a statin-treated patient does not predict plaque composition as it does for a statin-naive patient with a similar score. A CAC score is an important decision tool, and CAC testing should be performed to help guide patient-clinician decisions. However, without considering when it is too early, too late to be useful, or too often (eg, yearly), an opportunity is missed to use this important tool selectively, focusing on those who would most benefit.

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