NLA Scientific Statement on CAC: CAC-guided treatment recommendations and other considerations
Outline

1. Approach to the patient with CAC=0
2. Treatment of those with a high CAC score, multi-vessel CAC, or left main CAC
3. CAC in familial hypercholesterolemia
4. CAC in diabetes and metabolic syndrome
5. CAC and allocation of aspirin and anti-hypertensive therapies
6. Other consideration: CAC in those on statins
7. Other consideration: Repeat CAC testing
Part 1 - Section 4 of NLA CAC Statement

APPROACH TO THE PATIENT WITH CAC=0
Conventional view of risk factors

Concept of negative risk factors
Comparing “Negative Risk Markers” in the Multi-Ethnic Study of Atherosclerosis

- A CAC score of zero is the strongest “negative risk factor” for the development of ASCVD.
- Imaging Hypothesis – due to superior sensitivity, imaging tests for subclinical atherosclerosis are excellent at “ruling out” or “downgrading” risk estimates.

FIGURE 4. Relationship between pre-test and post-test cardiovascular disease risk after the knowledge of the negative result of each risk marker. ABI, ankle brachial index; BNP, B-type natriuretic peptide; CAC, coronary artery calcium; CHD, coronary heart disease; cIMT, carotid intima-media thickness; hsCRP, high-sensitivity C-reactive protein. Reprinted with permission from Blaha et al. [46].

In intermediate-risk or selected borderline-risk adults, if the decision about statin use remains uncertain, it is reasonable to use a CAC score in the decision to withhold, postpone or initiate statin therapy.
Key points

• CAC=0 is associated with highly favorable cardiovascular and non-cardiovascular prognosis. CAC=0 is the strongest “negative risk marker” for ASCVD.
• In the absence diabetes mellitus, active cigarette smoking or a family history of premature ASCVD, statin therapy in those with CAC=0 is associated with limited expected benefit.
• The absolute ASCVD risk reduction with statin therapy is proportional to the CAC score.

Recommendations

• In adults 40-75 years of age with LDL-C 70-189 mg/dL and without diabetes, active cigarette smoking or a strong family history of premature ASCVD, it is reasonable to defer statin initiation in those with CAC=0 (COR IIa, B-NR)

• In adults age 76-80 years of age in whom the decision about initiation of statin therapy is uncertain, it is reasonable to use CAC=0 as a factor favoring deferral or avoidance of statin therapy (COR IIb, B-NR)
TREATMENT OF THOSE WITH A HIGH CAC SCORE, MULTI-VESSEL CAC, OR LEFT MAIN CAC
Continuum of ASCVD Risk

Primary Prevention → Advanced Subclinical Atherosclerosis? → Secondary Prevention

Blaha MJ, AJC 2016
Primary prevention patients with extensive CAC (CAC ≥1,000) are unique in their burden of coronary and extra-coronary disease and in their long-term outcomes. Those with CAC ≥1,000 can be found on imaging to have a dispersed pattern of calcification in their coronary artery tree (the majority with 4-vessel disease) and diffuse extra-coronary calcification (TAC, AVC, and MVC). In addition, their annualized CVD mortality rates exceed those of high-risk secondary prevention patients from the FOURIER trial (0.8%/year vs. 0.7%/year). AVC = aortic valve calcium; CAC = coronary artery calcium; CVD = cardiovascular disease; MVC = mitral valve artery; TAC = thoracic artery calcium.
Agatston Score = 200
Area of CAC = 50 mm\(^2\)
Mean Density = 450 HU (weighting factor = 4)
Number of Vessels = 1
Pattern = Concentrated
Number of Lesions = 2
Lesion Type = Large

Agatston Score = 200
Area of CAC = 100 mm\(^2\)
Mean Density = 232 HU (weighting factor = 2)
Number of Vessels = 4
Pattern = Diffuse
Number of Lesions = 8
Lesion Type = Small
Left Main calcium is independently associated with cardiovascular-specific and total mortality beyond the total calcium score: The Coronary Artery Calcium Consortium

28,147 participants with CAC >0
21.7% with Left Main CAC

40% increased risk of CVD and total mortality
Key points

• For a given CAC score, a diffuse distribution of CAC suggests higher risk than more localized CAC.
• The presence of left main coronary calcification, especially when >25% of the total score is in the left main, suggests higher risk.
• There is no evidence to support the benefit of performing stress testing, or invasive coronary arteriography in asymptomatic individuals with high coronary calcium scores.
• A CAC score ≥100 is associated with >7.5% 10-year ASCVD risk, the guideline-based threshold of statin benefit in primary prevention.
• A CAC score ≥300 is associated with proportionately higher ASCVD risk than those with scores >100, a finding suggesting benefit from greater LDL-C lowering.
• A CAC score ≥1000 is associated with an annual risk similar to that of the placebo group in the FOURIER trial, a finding consistent with the potential value of very aggressive LDL-C lowering along with other ASCVD risk reduction strategies.
Recommendations

• In adults with predominant left main coronary calcification, multi-vessel coronary involvement, or a high CAC score, stress testing or invasive coronary arteriography, in the absence of clinically relevant symptoms, is not recommended. (COR III-Harm)

• In adults with CAC scores ≥ 100, initiation of statin therapy is reasonable. (COR IIa, LOE B-NR)

• In adults with CAC scores ≥300, and especially in those with CAC scores ≥ 1000, it is reasonable to use high intensity statin therapy, and if necessary, guideline-based add-on LDL-C lowering therapies to achieve a ≥50% reduction in LDL-C, and optimally and LDL-C <70 mg/dL is reasonable. (COR IIa, LOE C-LD).
Part 3 - Section 7 of NLA CAC Statement

CAC IN FAMILIAL HYPERCHOLESTEROLEMIA
Coronary Artery Calcium and Cardiovascular Events in Patients With Familial Hypercholesterolemia Receiving Standard Lipid-Lowering Therapy

Marcio H. Miname, MD, PhD, a Marcio Sommer Bittencourt, MD, PhD, MPH, b,c Sérgio R. Moraes, BSc, a Rômulo L.M. Alves, BSc, a Pamela R.S. Silva, PhD, a Cinthia E. James, PhD, a Alexandre C. Pereira, MD, PhD, a José E. Krieger, MD, PhD, a Khurram Nasir, MD, MSc, MPH, a Raul D. Santos, MD, PhD a,b

OBJECTIVES The aim of this study was to evaluate the role of coronary artery calcium (CAC) as a predictor of atherosclerotic cardiovascular disease (ASCVD) (fatal or not myocardial infarction, stroke, unstable angina requiring revascularization, and elective myocardial revascularization) events in asymptomatic primary prevention molecularly proven heterozygous familial hypercholesterolemia (FH) subjects receiving standard lipid-lowering therapy.

Baseline LDL-C (mg/dl)  269 ± 70
Baseline HDL-C (mg/dl)  48 ± 13
Baseline triglycerides (mg/dl)  136 ± 66
Baseline cholesterol-years score  11,633 (7,330)
On-treatment LDL-C (mg/dl)  150 ± 56
Glucose  106 ± 39
LDL R mutations:
Null  99 (48.3%)
Defective  88 (42.9%)
Not classified  18 (8.8%)
Baseline statin use  142 (68.9%)
Statin use at follow-up  199 (96.6%)
High-dose statin use at follow-up  164 (79.6%)
Ezetimibe use at follow-up  130 (64.0%)

CONCLUSIONS CAC was independently associated with ASCVD events in patients with FH receiving standard lipid-lowering therapy. This may help further stratify near-term risk in patients who might be candidates for further treatment with newer therapies. (J Am Coll Cardiol Img 2019;12:1797-804) © 2019 by the American College of Cardiology Foundation.
Clinical significance of zero coronary artery calcium in individuals with LDL cholesterol ≥ 190 mg/dL: The Multi-Ethnic Study of Atherosclerosis

Pratik B. Sandesara a,*, Anurag Mehta a, Wesley T. O'Neal a, Heval M. Kelli a, Vasanth Sathiyakumar c, Seth S. Martin c, Michael J. Blaha c, Roger S. Blumenthal c, Laurence S. Sperling a,b

Risk of future CHD, CVD, and mortality associated with CAC = 0 in those with LDL-C ≥ 190 mg/dL (N = 246) c.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Events</th>
<th>Events per 1000 person-years</th>
<th>10-year risk</th>
<th>Risk/year</th>
<th>HR b (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD</td>
<td>CAC &gt; 0</td>
<td>29</td>
<td>18.6</td>
<td>17%</td>
<td>1.7%</td>
</tr>
<tr>
<td></td>
<td>CAC = 0</td>
<td>4</td>
<td>3.8</td>
<td>3.7%</td>
<td>0.4%</td>
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<tr>
<td>CVD</td>
<td>CAC &gt; 0</td>
<td>40</td>
<td>26.4</td>
<td>20%</td>
<td>2.0%</td>
</tr>
<tr>
<td></td>
<td>CAC = 0</td>
<td>5</td>
<td>4.7</td>
<td>3.7%</td>
<td>0.4%</td>
</tr>
</tbody>
</table>
Absence of Coronary Artery Calcification in Middle-Aged Familial Hypercholesterolemia Patients Without Atherosclerotic Cardiovascular Disease

included. Seven studies representing 93% of the pooled population involved the use of genetic sequencing to provide a molecular diagnosis of HeFH; the other 2 studies utilized clinical criteria such as the US MEDPED (Make Early Diagnosis to Prevent Early Death) and Simon-Broome diagnostic criteria.
Recommendations

• In selected adults with severe primary hypercholesterolemia, in the absence of extreme LDL-C elevation, additional major ASCVD risk factors or a family history of premature ASCVD, CAC scoring may be reasonable to inform decision-making about the need for add-on therapy to maximally tolerated statins. (COR IIb, C-LD).

• In adults with severe primary hypercholesterolemia and CAC>0, heightened ASCVD risk status is confirmed, favoring more aggressive, guideline based LDL-C lowering. (COR IIa, C-LD)
Part 4 - Section 8 of NLA CAC Statement

CAC IN DIABETES AND METABOLIC SYNDROME
Coronary Artery Calcium Score for Long-term Risk Classification in Individuals With Type 2 Diabetes and Metabolic Syndrome From the Multi-Ethnic Study of Atherosclerosis

Shaista Malik, MD, PhD, MPH; Yanglu Zhao, MD, MS; Matthew Budoff, MD; Khurram Nasir, MD; Roger S. Blumenthal, MD; Alain G. Bertoni, MD, MPH; Nathan D. Wong, PhD, MPH

A Coronary heart disease

B Atherosclerotic cardiovascular disease
CAC Stratifies Risk in Type 2 Diabetes: Independent of Diabetes Duration and HbA1c

No. at risk
CAC score of 0 and diabetes duration <10 y 151 141 131 121 118 47
CAC score of 0 and diabetes duration ≥10 y 64 60 54 52 47 15
CAC score of 1-399 and diabetes duration <10 y 149 141 131 123 110 35
CAC score of 1-399 and diabetes duration ≥10 y 105 95 84 78 72 28
CAC score of ≥400 and diabetes duration <10 y 54 47 42 40 34 12
CAC score of ≥400 and diabetes duration ≥10 y 52 44 36 27 22 7
CAC Stratifies CHD and CVD Risk in Type 2 Diabetes: Independent of Insulin Use
The Association of Coronary Artery Calcification With Subsequent Incidence of Cardiovascular Disease in Type 1 Diabetes

The DCCT/EDIC Trials

Matthew Budoff, MD, Jye-Yu C. Backlund, MPH, David A. Bluemke, MD, PhD, Joseph Polak, MD, MPH, Ionut Bebu, PhD, David Schade, MD, Suzanne Strowig, MSN, Philip Raskin, MD, John M. Lachin, ScD, for the DCCT/EDIC Research Group

Table 3: Association Between CAC at EDIC Years 7 to 9 and Subsequent CVD and MACE Among Those Still at Risk During the EDIC Study

<table>
<thead>
<tr>
<th>Subclinical CAC Score (Agatston Units)</th>
<th>CVD (n = 1,158)</th>
<th>MACE (n = 1,187)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hazard Ratio</td>
<td>p Value</td>
</tr>
<tr>
<td>Model A†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>1.00 (Reference)</td>
<td>1.00 (Reference)</td>
</tr>
<tr>
<td>&gt;0-100</td>
<td>1.17 (1.02-2.38)</td>
<td>0.0415</td>
</tr>
<tr>
<td>&gt;100-300</td>
<td>4.17 (2.23-7.80)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>&gt;300</td>
<td>6.06 (3.22-11.40)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Chi-square test†</td>
<td>38.68</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Model B‡</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>1.00 (Reference)</td>
<td>1.00 (Reference)</td>
</tr>
<tr>
<td>&gt;0-100</td>
<td>1.54 (0.91-2.60)</td>
<td>-0.1060</td>
</tr>
<tr>
<td>&gt;100-300</td>
<td>4.05 (2.14-7.64)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>&gt;300</td>
<td>4.73 (2.47-9.08)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Chi-square test‡</td>
<td>30.53</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Values in bold indicate statistically significant findings. *Adjusted for scanning site, sex, age, systolic blood pressure, antihypertensive medication, LDL, HDL, and smoking at EDIC years 7 to 9, and DCCT baseline family history of MI. †Adjusted for scanning site, sex, study cohort, log mean HbA1c, age, systolic blood pressure, antihypertensive medication, LDL, HDL, and smoking at EDIC years 7 to 9, and DCCT baseline family history of MI. MI = myocardial infarction; other abbreviations as in Tables 1 and 2.
Recommendations

• In adults 40-75 years of age with type 2 diabetes and an LDL-C 70-189, a moderate or high intensity statin is indicated, regardless of CAC score. (COR I, LOE A)

• In adults 40-75 years of age with type 2 diabetes age in whom the decision has been made to initiate statin therapy, it is reasonable, for those with a CAC score >100, to choose a high intensity statin. (COR IIa, LOE C-LD)

• In adults 30-39 years of age with long-standing diabetes* and risk factors or microangiopathy, CAC scoring may be reasonable to aid in ASCVD risk stratification and statin treatment shared decision making. (COR IIb, LOE C-LD)

• In adults older than 75 years of age with type 2 diabetes, in whom the decision to employ a statin for primary prevention is uncertain, CAC scoring is reasonable to aid in statin treatment shared decision making (COR IIa, LOE C-LD).

*type 1 diabetes of ≥20 years duration or type 2 diabetes of ≥10 years duration
CAC AND ALLOCATION OF ASPIRIN AND ANTI-HYPERTENSIVE THERAPIES

Part 5 - Section 11 of NLA CAC Statement
Aspirin Net Benefit According to CAC Scores – Updated 2020 Analysis

Cainzos-Achirica, et al.– Circulation 2020
Conclusions—Combined CAC-imaging and assessment of global ASCVD risk has potential to guide personalized SBP goals (e.g., choosing a traditional goal of 140 or a more intensive goal of 120 mmHg), particularly among adults with estimated ASCVD risk 5-15% and pre-hypertension or mild hypertension.
Role of Coronary Artery Calcium for Stratifying Cardiovascular Risk in Adults With Hypertension
The Coronary Artery Calcium Consortium

Figure 2. Coronary artery calcium (CAC) score equivalent of SPRINT (Systolic Blood Pressure Intervention Trial)-level risk among participants age >50 y. Graph shows the annual cardiovascular disease (CVD) mortality rate as a function of CAC scores among hypertensive patients age >50y. Horizontal red line represents the age-adjusted CVD death rate observed in the SPRINT trial (0.35%/y). These lines intersect at CAC=270, with lower limit of confidence (accounting for possible 15% underestimation of risk in the CAC Consortium) at CAC=166.

Uddin et al. *Hypertension*. 2019
Key points

• CAC appears to reclassify risk in patients with Stage 1 hypertension, and may be useful for guiding decisions about pharmacotherapy.

• A CAC score 220 appears to identify patients with annual ASCVD risk similar to those enrolled in the Systolic Blood Pressure Intervention Trial (SPRINT). CAC may be useful in guiding blood pressure targets.
CAC IN THOSE ON STATINS
Effects of Statins on Coronary Atherosclerotic Plaques

The PARADIGM (Progression of Atherosclerotic Plaque Determined by Computed TomoGraphic Angiography Imaging) Study

Sang-Eun Lee, MD, PhD,a,b Hyuk-Jae Chang, MD, PhD,a,b Ji Min Sung, PhD,a,b Hyung-Bok Park, MD,b,c Ran Heo, MD,b,d Asim Rizvi, MD,e Fay Y. Lin, MD,e Amit Kumar, MSc,e Martin Hadamitzky, MD,f Yong Jin Kim, MD, PhD,3 Edoardo Conte, MD,g Daniele Andreini, MD, PhD,3 Gianluca Pontone, MD, PhD,3 Matthew J. Budoff, MD,3 Ilan Gottlieb, MD, PhD,3 Byoung Kwon Lee, MD, PhD,3 Eun Ju Chun, MD, PhD,3 Filippo Cademartiri, MD, PhD,3 Erica Maffei, MD,3 Hugo Marques, MD,3 Jonathon A. Leipsic, MD,3 Sanghoon Shin, MD,3 Jung Hyun Choi, MD, PhD,3 Kavitha Chinnaian, MD,3 Gilbert Raff, MD,3 Renu Virmani, MD,3 Habib Samady, MD,3 Peter H. Stone, MD,3 Daniel S. Berman, MD,3 Jagat Narula, MD, PhD,3 Leslee J. Shaw, PhD,3 Jeroen J. Bax, MD, PhD,3 James K. Min, MD,e

A

Statin-naive patients
Baseline Follow-up
Statin-taking patients
Baseline Follow-up

Plaque composition
Dense calcium Fibrous
Low-attenuation Fibro-fatty

B

Annual PAV Change (%/year)
Overall
Calcified
Noncalcified

p = 0.002
p < 0.001
p < 0.001

No Statin Statin
CAC – Predicts risk similarly in statin naïve and statin treated

<table>
<thead>
<tr>
<th>Cohort characteristics</th>
<th>NOT ON STATIN THERAPY</th>
<th>ON STATIN THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>21,874</td>
<td>6,151</td>
</tr>
<tr>
<td>Mean age</td>
<td>54</td>
<td>57</td>
</tr>
<tr>
<td>Women</td>
<td>38%</td>
<td>28%</td>
</tr>
<tr>
<td>White</td>
<td>95%</td>
<td>94%</td>
</tr>
<tr>
<td>Mean CAC score</td>
<td>107 +/- 332</td>
<td>281 +/- 664</td>
</tr>
</tbody>
</table>

**Mortality event rates (per 1000 person-years) and Hazard ratios**

<table>
<thead>
<tr>
<th>CHD Mortality</th>
<th>NOT ON STATIN THERAPY</th>
<th>ON STATIN THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAC 0</td>
<td>0.1</td>
<td>Reference</td>
</tr>
<tr>
<td>CAC 1-99</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>CAC 100-399</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>CAC ≥400</td>
<td>1.9</td>
<td>2.5</td>
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</table>

<table>
<thead>
<tr>
<th>CVD Mortality</th>
<th>NOT ON STATIN THERAPY</th>
<th>ON STATIN THERAPY</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAC 0</td>
<td>0.3</td>
<td>Reference</td>
</tr>
<tr>
<td>CAC 1-99</td>
<td>0.8</td>
<td>1.1</td>
</tr>
<tr>
<td>CAC 100-399</td>
<td>1.2</td>
<td>1.7</td>
</tr>
<tr>
<td>CAC ≥400</td>
<td>4.0</td>
<td>3.9</td>
</tr>
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</table>

**Association of CAC components with CHD and CVD mortality among participants with CAC >0**

<table>
<thead>
<tr>
<th>CHD Mortality</th>
<th>Age and sex + volume OR density score adjusted</th>
<th>Age and sex + volume OR density score adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ln (Volume score), per SD</td>
<td>2.3 (1.6, 3.1)</td>
<td>2.5 (1.6, 3.8)</td>
</tr>
<tr>
<td>Density score, per SD</td>
<td>0.69 (0.49, 0.95)</td>
<td>1.1 (0.7, 2.0)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CVD Mortality</th>
<th>Age and sex + volume OR density score adjusted</th>
<th>Age and sex + volume OR density score adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ln (Volume score), per SD</td>
<td>1.8 (1.4, 2.2)</td>
<td>1.9 (1.4, 2.6)</td>
</tr>
<tr>
<td>Density score, per SD</td>
<td>0.78 (0.63, 0.97)</td>
<td>0.9 (0.6, 1.3)</td>
</tr>
</tbody>
</table>

Osei et al. Atherosclerosis. 2020
Key points

• Statins delipidate plaque, decreasing volume of non-calcified plaque and increasing volume of calcified plaque.

• CAC remains a strong risk predictor in statin treated patients, similar to risk discrimination observed in statin naïve patients.
Part 7 - Section 9 of NLA CAC Statement

REPEAT CAC TESTING
CAC=0: Time to Rescan
(Warranty Period)

Dzaye et al. JACC. 2020
Progression of Coronary Calcium and Incident Coronary Heart Disease Events

MESA (Multi-Ethnic Study of Atherosclerosis)

Matthew J. Budoff, MD,* Rebekah Young, PhD,† Victor A. Lopez, MS,‡ Richard A. Kronmal, PhD,†
Khurram Nasir, MD, MPH,*§‖ Roger S. Blumenthal, MD,§ Robert C. Detrano, MD, PhD,‡
Diane E. Bild, MD, MPH,*** Alan D. Guerci, MD,†† Kiang Liu, PhD,‡‡ Steven Shea, MD,§§
Moyses Szklar, MD,‖‖ Wendy Post, MD,§ Joao Lima, MD,§ Alain Bercutini, MD, MPH,¶¶
Nathan D. Wong, PhD, MPH‡‡

<table>
<thead>
<tr>
<th>Adjusted percentage Δ in CAC/yr*</th>
<th>Total CHD [Model: 283/2,874]</th>
<th>Hard CHD [Model: 167/2,874]</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5%</td>
<td>Reference</td>
<td>Reference</td>
</tr>
<tr>
<td>5–14%</td>
<td>1.1 (0.7–1.8)</td>
<td>1.0 (0.5–1.9)</td>
</tr>
<tr>
<td>15–29%</td>
<td>1.6 (1.0–2.5)</td>
<td>1.4 (0.8–2.6)</td>
</tr>
<tr>
<td>≥30%</td>
<td>1.5 (0.9–2.4)</td>
<td>1.4 (0.7–2.8)</td>
</tr>
</tbody>
</table>
Key points

• Recommended timing for repeat CAC scoring depends upon the baseline ASCVD risk of the individual, varying from 3 to 7 years.

• CAC scores increase by approximately 20-25% per year. Hence, the Agatston score generally increases exponentially, with scores in the range of 0-100 providing the greatest risk discrimination compared to higher scores.

• CAC should be measured only if such measurement will change treatment decisions.

• CAC does not regress.

• CAC progression cannot be used to measure the efficacy of statin therapy (statins modestly increase the CAC score). Statins delipidate plaque, decreasing volume of calcified plaque.

• In individuals who undergo repeat CAC scoring, progression of >20-25% per year or an increase to a score of ≥400 in an individual with a previous CAC score >0 is consistent with accelerated ASCVD progression.
Recommendations

• In adults with CAC=0, it is reasonable to repeat CAC scoring at the following intervals:
  
  Low risk (<5% 10 year risk): 5-7 years  
  Borderline to intermediate risk (5-19.9% 10 year risk): 3-5 years  
  High risk or diabetes: 3 years  
  (Class IIa, B-NR)

• In adults with CAC scores 1-99, it may be reasonable to repeat CAC scoring in 3-5 years if the results might change treatment decisions (Class IIb LOE B-NR).

• In adults with CAC scores ≥100 and an LDL-C ≥70 mg/dL, repeat CAC scoring at 3 years may be reasonable to assess for accelerated progression (>20-25% per year) and/or an increase to a CAC score >300, findings that may favor more aggressive LDL-C lowering. (Class IIb, LOE C-LD).
Other topics covered

Future of CAC Scoring

1. Is CAC scoring cost-effective?
2. Are there clinical outcomes trials with CAC as entry criteria supporting the use of CAC?
3. Is CAC typically covered by insurance?
4. What is the future of CAC score reporting?
5. What is the relationship of polygenic risk scores with CAC?
6. What new tools facilitating CAC-based risk communication are being developed?