Advances In Nuclear Cardiology and Cardiac CT: New Developments in PET and SPECT

How Does Coronary Artery Calcium Scoring Fit Into A Preventive Strategy

No Disclosures

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CACS: How Does It Fit In Prevention
Assumptions For “Incremental Value”

- Minimal Procedural Risk
- Diagnosis of early atherosclerosis
- Accurate Risk Stratification Among Heterogeneous Populations at Risk (Reclassify risk)
- Change behavior and modify risk factors accordingly – identifying CAD and defining risk is not enough
- Target appropriate therapy based on test results
- Reduce CHD events through targeted therapeutic and lifestyle interventions
- Save downstream costs --or not have costs increase!
Coronary Artery Atherosclerotic Plaque Area vs CAC (EBCT)
Individual Coronary Arteries


CACS vs. CT Plaque Morphology
Asymptomatic Subjects

1043 Asymptomatic South Koreans (35-75 years old) without prior history of CAD or renal insufficiency

CACS =0
866 (83%) Patients
40 (4.6%) with exclusively NCP
10 (1.2%): >50% stenosis
5 (0.6%): >75% stenosis

Coronary Atherosclerosis by CTA: CONFIRM
Symptomatic Patients

3.5%>50%
1.4%>70%

10,037 Symptomatic patients at intermediate risk for CAD (~43%) who had CTA and CACS
51% with CACS=0

Villines et al JACC 2011;58:2533

CACS And Coronary Atherosclerosis

52 year old Male with exertional SOB; hypertension; TC=196, LDL=133
CACS= 224
PEDTHO
Cardiac Evaluation for Chest Pain Symptoms

- 58 year old man with multiple risk factors for CAD, referred for evaluation of C.P – probable angina
- ATP III Risk Score: 22% (High)/ Intermediate Pretest Likelihood
- The patient had an exercise myocardial perfusion SPECT to further evaluate
- Exercise stress test results
  - Exercise time: 8 minutes, 7 seconds Bruce protocol
  - Maximal HR: 160 (100% predicted)
  - Maximal BP: 154/50 mmHg
  - Symptoms: None
  - Upsloping ST-segment depression: 1 mm at peak exercise which resolved 30 seconds into recovery.
  - Duke Treadmill Score: 2 (intermediate)

PEDTHO
Exercise SPECT Results
**PEDTHO**
CTA In a Patient With Continuing C.P.

<table>
<thead>
<tr>
<th>Right</th>
<th>Circumflex</th>
<th>First Obtuse Marginal</th>
</tr>
</thead>
</table>

**CACS = 0**

**PEDTHO**
CT vs. Invasive Angiography

*Left Main Stenosis*
Risk Stratification: Relative Risk CACS 0 vs. CACS>0
Meta-analysis of 71,595 Asymptomatic Patients

Raw CHD Event Rates (CD,NFMI, Rev): CACS=0 0.47% (0.1%/year) vs.
CACS>0 4.14% (1.0%/year)
Mean follow-up: 4.2 years

<table>
<thead>
<tr>
<th>Study</th>
<th>Point</th>
<th>Lower CI</th>
<th>Upper CI</th>
<th>Relative Risk Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CACS=0</td>
<td>0.05</td>
<td>0.001</td>
<td>0.25</td>
<td></td>
</tr>
<tr>
<td>Raggi (2001)</td>
<td>0.04</td>
<td>0.01</td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td>Wong (2002)</td>
<td>0.10</td>
<td>0.04</td>
<td>0.25</td>
<td></td>
</tr>
<tr>
<td>Kondos (2003)</td>
<td>0.10</td>
<td>0.05</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>Shear (2003)</td>
<td>0.13</td>
<td>0.07</td>
<td>0.23</td>
<td></td>
</tr>
<tr>
<td>Shemesh (2004)</td>
<td>0.15</td>
<td>0.09</td>
<td>0.25</td>
<td></td>
</tr>
<tr>
<td>Greenland (2004)</td>
<td>0.18</td>
<td>0.12</td>
<td>0.29</td>
<td></td>
</tr>
<tr>
<td>Arad (2006)</td>
<td>0.18</td>
<td>0.14</td>
<td>0.22</td>
<td></td>
</tr>
<tr>
<td>LaMonte (2005)</td>
<td>0.18</td>
<td>0.12</td>
<td>0.26</td>
<td></td>
</tr>
<tr>
<td>Taylor (2005)</td>
<td>0.17</td>
<td>0.11</td>
<td>0.25</td>
<td></td>
</tr>
<tr>
<td>Budoff (2007)</td>
<td>0.16</td>
<td>0.11</td>
<td>0.23</td>
<td></td>
</tr>
<tr>
<td>Becker (2008)</td>
<td>0.15</td>
<td>0.11</td>
<td>0.22</td>
<td></td>
</tr>
<tr>
<td>Defranco (2008)</td>
<td>0.15</td>
<td>0.11</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>Summary Risk</td>
<td>0.15</td>
<td>0.11</td>
<td>0.21</td>
<td></td>
</tr>
</tbody>
</table>

CACS of 0 in 41% of patients

Sarwar A et al. J Am Coll Cardiol Img 2009;2:675-688

CACS For Defining Low Risk All-Cause Mortality Implications in Clinical Decision-making

44,052 Asymptomatic individuals mean age 54 yrs, without CHD referred for CACS study
Mean followup 5.6 years

Men (54% of subjects) Women (46% of subjects)

The value of a CACS of Zero!

All cause mortality 1000 person-years
CACS=0 0.87 (0.72-1.05)
CACS1-10 1.32 (1.48-2.48)
CACS >10 7.48 (6.92-8.04)

“In non-high risk patients, the absence of CAC could be used as a rationale to emphasize lifestyle changes, scale back on costly preventative pharmacotherapy and refrain from frequent cardiac imaging”

### Warranty Period For a Normal CACS of 0

- **422 subjects with CACS=0 who had annual scans up to 5 years.**
  - 25% with CAC at 4.1±0.9 years (6.1%/year)

- **2948 subjects with CACS=0 who had serial imaging at mean 2.4 years.**
  - 16.1% with CAC at 2.4 years (6.6%/year)

Conversion associated with age>40, diabetes and smoking (all p<0.001) with a mean CACS at the time of conversion of 19±19

Min et al JACC 2010;55:1110

*Kronmal et al. Circ 2007;115:2722

### Defining High Risk: CACS And Cardiac Events Among Different Ethnic Groups

**MESA: 6722 Patients**

<table>
<thead>
<tr>
<th>AMI and Cardiac Death</th>
<th>Any Coronary Event</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Coronary artery calcium score</strong></td>
<td></td>
</tr>
<tr>
<td>&gt;300</td>
<td>&gt;101-300</td>
</tr>
<tr>
<td>1-100</td>
<td>0</td>
</tr>
</tbody>
</table>

A similar increase in risk among all ethnic groups

CACS added significantly to traditional RF’s for predicting cardiac events and in all ethnic groups ---- *Adds to >70,000 patients worth of prognostic data*

Risk Stratification: CACS and Survival
Diabetics vs Non-Diabetics

Non Diabetics (N=9,474) Diabetics (N=903)

Cumulative Survival

0-10 (n=5600)
11-100 (n=1854)
101-400 (n=1251)
401-1000 (n=508)
>1000 (n=251)

Follow-up (Years)


Risk Stratification: CACS Testing In The Elderly*

CHD Event Free Survival CVD Event Free Survival

*Mean age 71 years, range 62 to 85

CACS In the Asymptomatic Patient
LANJOH

• 51 year old asymptomatic man: Low Pretest Probability CAD

• Risk factors for CAD:
  Male gender
  Hypertension controlled with medication
  Hyperlipidemia (total chol 230/LDL 182/HDL 32)
  Previous history cigarette smoking

• Medications:
  Cardizem CD 300 mg q Day
  Zocor 10 mg q PM

• Poor compliance with dietary prescription
• No routine physical activity

ATP III Risk Score: 26% (High)

LANJOH - CT Results

April 2000
CACS: 780
LANJOH - SPECT Results

- ETT Time: 11.0 minutes
- Maximal HR: 168 BPM (100% target)
- METS: 12.9
- Symptoms: Fatigue
- DTS: 11 (low risk)
- LVEF = 58%

LANJOH - Angiographic Results

- Right Coronary Artery
- LAD Coronary Artery
**LANJOH - SPECT Results**

Integrating SPECT *After CACS* To Assess Risk
Detecting *Asymptomatic* Ischemia

<table>
<thead>
<tr>
<th>% Pts</th>
<th>Pts. With Metabolic Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;100</td>
<td>1.5%</td>
</tr>
<tr>
<td>101-399</td>
<td>11.3%</td>
</tr>
<tr>
<td>&gt;400</td>
<td>13%</td>
</tr>
</tbody>
</table>

- N = 657 Overall 1.7%
- N = 602 Overall 10%
- N = 669 Overall 26%

*He et al (N=411)*
*Anand et al (N=220)*
*Moser (N=102)*
*Berman (N=1195)*

~10% of subjects
Risk Stratification
Reclassifying Risk: Why CACS?

- Framingham Risk Score

  *Indirect population based analysis*

  Predicts the probability that patients will develop coronary atherosclerosis and manifest CHD over a 10 year horizon based on a limited number of known risk factors.

- CACS

  *Direct individual patient analysis*

  Identifies the presence and extent of coronary atherosclerosis in an individual patient and predicts outcome based on severity.

CACS and Framingham Risk Score
Reclassifying Risk to Guide Clinical Decision-Making

1029 non-diabetic subjects, 90% men, age 65.7 yrs. followed for 7.0 years (median)

“A high CACS can significantly modify predicted risk and thereby alter clinical decision making, especially for those in the intermediate-risk category (62% of patients) for whom decision making is most uncertain.”

* *p <0.001 vs. all other CAC scores
+ p=0.01 vs. CACS=0

Greenland et al. JAMA 2004; 291: 210
CACS For Clarifying Risk in Subjects At Intermediate Framingham Risk: A Meta-Analysis

Annual Risk of CHD Death or AMI

Tertile 1
CACS 0-99

Tertile II
CACS 100-399

Tertile III
CACS =400

<Low Risk FRS 0.4%

1.3%

2.4%

>High Risk FRS

Arad et al JACC 2005;46:158
Vliegenthart et al Circulation 2005;112:572
LaMonte et al Am J Epidemiology 2005;162:421

Greenland et al JAMA 2004;291:210

Framingham Risk Score vs. CACS Predicting Outcome: EISNER

Shaw et al JACC 2009 54: 1258-1267
Reclassifying Clinical Risk with CACS

44,052 patients, without CAD, median follow-up 5.6 year for death
Risk factors: Smoking, hyperlipidemia, DM, hypertension, +family hx.

Nasir K et al. Circ. CV Imaging 2012; 5:467
Screening for CAD Asymptomatic Patients

- CT Coronary Artery Calcium Scoring: Appropriateness Use Criteria
  
  JACC 2010;56:1864

  Low CHD Risk but
  
  Family History Premature CHD: A (7)
  Intermediate CHD Risk: A (7)
  High CHD Risk: U (4)

CACS After Exercise SPECT: SACALE

65 year old M.D. with hypertension, hyperlipidemia, cigarette smoking and recent onset atypical chest pain

CAD Probability: Intermediate

ATPIII Risk Score: 28% (High)

ETT Results:
Exercise Time: 8.5 min
Maximal HR: 125 bpm (80% target)
Asymptomatic
No ST changes
Duke TS: 8.5 (low risk)
CT CACS Following A Normal SPECT

CACS = 740

Enhanced Risk Stratification
CACS After SPECT In Patients Without CAD

- Rosanski et al. JACC 2007; 49:1352
- Schenker et al. Circulation 2008; 117:1693
- Chang et al. JACC 2009; 54:1872
Enhanced Risk Stratification In Diabetics Combining CT CACS and SPECT

<table>
<thead>
<tr>
<th>% of Myocardium</th>
<th>CAC 0–100</th>
<th>CAC 101–400</th>
<th>CAC 401–1000</th>
<th>CAC &gt;1000</th>
</tr>
</thead>
<tbody>
<tr>
<td>0%</td>
<td>100%</td>
<td>98%</td>
<td>96%</td>
<td>90%</td>
</tr>
<tr>
<td>1–5%</td>
<td>100%</td>
<td>92%</td>
<td>83%</td>
<td>77%</td>
</tr>
<tr>
<td>&gt;5%</td>
<td>100%</td>
<td>80%</td>
<td>64%</td>
<td>48%</td>
</tr>
</tbody>
</table>

RR=9.20 (1.48, 57.19), P=0.017

Interaction P=0.003 (unadjusted) and <0.0001 (adjusted for United Kingdom Prospective Diabetes Study risk score [Stevens RJ et al. Clin Sci 2001;101:671-679]). Event-free survival estimates are from a stratified Cox model

p = 0.003

Anand et al Eur Heart J 2006;27:713-721

Improved Risk Stratification: Combining CACS With SPECT

1126 patients followed up to 12 years (median 6.9), 84% FRS> Intermediate

Patients With Normal SPECT

Log rank p <0.001

Time point analysis:
P value 0.03 at year 3

Impact of CACS Results
Behavioral Lifestyle Changes

- 703 asymptomatic men and women without CAD self-referred for calcium scoring (age 53.7); 1 year followup
- 393 CACS>0 (55%) 59% men; 43% women

Behavioral Changes CACS>0
- Physician consultation: RR 1.61, p=.01
- New ASA usage: RR 1.86, p<.01
- New Cholesterol Meds: RR 3.54, p=.01
- Decreased dietary fat: RR 1.58, p=.02
- Increased worry: RR 2.73, p=.03

Wong et al. Am J Cardiol 1996;78:1220

CACS Results: Do They Alter Therapy

1640 asymptomatic men, active army duty, 40-50 years old, 10-year FRS 4.6 (2.6)
CACS of 0: 1263 (77.6%); TChol: 204 (36); LDL:128 (32); HDL: 50 (13) mg/dl

Statin Use Based On CACS Results
Aspirin Use Based On CACS Results

Taylor AJ et al. J Am Coll Cardiol 2008;51:1337
CACS With Normal PET MPI
Therapeutic Considerations

- 760 consecutive patients without known CAD who had CACS and normal Rb-82 PET

**CACS Distribution**

- Low FR Score
  - 30%
- Intermediate FR Score
  - 26%
- High FR Score
  - 38%

- Bybee KA et al JNC 2009

---

**Change in Therapy based on CACS**

760 consecutive patients without prior CAD who had CACS and normal Rb-82 PET

<table>
<thead>
<tr>
<th>Change in:</th>
<th>Agatston score range</th>
<th>Score = 0 n = 273</th>
<th>0 &lt; Score &lt; 400 n = 378</th>
<th>Score &gt; 400 n = 109</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any medication*</td>
<td></td>
<td>84 (34.3%)</td>
<td>174 (49.9%)</td>
<td>66 (65.4%)</td>
<td>.001</td>
</tr>
<tr>
<td>Statin</td>
<td></td>
<td>27 (11.0%)</td>
<td>65 (18.6%)</td>
<td>27 (26.7%)</td>
<td>.001</td>
</tr>
<tr>
<td>Non-statin cholesterol med</td>
<td></td>
<td>11 (4.5%)</td>
<td>11 (3.2%)</td>
<td>1 (1.0%)</td>
<td>.276</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td></td>
<td>19 (7.8%)</td>
<td>34 (9.7%)</td>
<td>16 (15.8%)</td>
<td>.072</td>
</tr>
<tr>
<td>ACE Inhibitor</td>
<td></td>
<td>15 (6.1%)</td>
<td>26 (7.5%)</td>
<td>14 (13.9%)</td>
<td>.048</td>
</tr>
<tr>
<td>Omega-3</td>
<td></td>
<td>20 (8.2%)</td>
<td>26 (7.5%)</td>
<td>10 (9.9%)</td>
<td>.726</td>
</tr>
<tr>
<td>Aspirin</td>
<td></td>
<td>34 (13.9%)</td>
<td>43 (12.3%)</td>
<td>16 (15.8%)</td>
<td>.632</td>
</tr>
<tr>
<td>Suggested OMT</td>
<td></td>
<td>9 (3.3%)</td>
<td>63 (16.7%)</td>
<td>24 (22.0%)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Bybee KA et al J NuclCardiol2009
Does Performing CACS Improve Patient Outcome Cost Effectiveness

CACS on Behavioral Changes, Risk Factor Modification and Downstream Costs

- EISNER: Prospective randomized trial: CACS +/- 2137 volunteers with 4 year f/u
- Results
  - Scan group: decrease in SBP, LDLC, waist circumference (p<0.50)
  - No increase in FRS vs. the no-scan group (p=0.003)
  - Increasing CACS related to improvements in SBP, DBP, TC, LDLC, Triglycerides, weight, FRS (p<0.001)
  - Downstream medical testing and costs comparable in the two groups: decreased costs and procedures in patients with CACS =0 (48%)/ CACS>400 (8%)
- No difference in events: annual death/mi event rate 0.4%

Rozanski et al JACC 2011;57:1622
Stress SPECT and ECHO

Invasive CA

Table 5: Estimated Annual Cardiovascular Procedural and Overall Costs in CAC Subgroups

<table>
<thead>
<tr>
<th>CAC Subgroups</th>
<th>% of Subjects</th>
<th>Procedural Costs</th>
<th>50th (25th, 75th)</th>
<th>% of Costs</th>
<th>Overall Costs</th>
<th>50th (25th, 75th)</th>
<th>% of Overall Costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10 (n = 779)</td>
<td>50.7%</td>
<td>$477 (65, $360)</td>
<td>$25 (15, $230)</td>
<td>18.5%</td>
<td>$477</td>
<td>$353 (445, $615)</td>
<td>20.0%</td>
</tr>
<tr>
<td>11-100 (n = 267)</td>
<td>21.0%</td>
<td>$444 (65, $360)</td>
<td>$35 (15, $230)</td>
<td>17.6%</td>
<td>$444</td>
<td>$353 (445, $615)</td>
<td>21.0%</td>
</tr>
<tr>
<td>101-399 (n = 287)</td>
<td>13.7%</td>
<td>$674 (65, $360)</td>
<td>$444 (65, $360)</td>
<td>22.6%</td>
<td>$674</td>
<td>$444 (65, $360)</td>
<td>22.6%</td>
</tr>
<tr>
<td>400-699 (n = 83)</td>
<td>6.5%</td>
<td>$1,088 (65, $360)</td>
<td>$552 (65, $360)</td>
<td>21.7%</td>
<td>$1,088</td>
<td>$552 (65, $360)</td>
<td>21.7%</td>
</tr>
<tr>
<td>700-1,000 (n = 31)</td>
<td>2.2%</td>
<td>$1,637 (65, $360)</td>
<td>$1,637 (65, $360)</td>
<td>25.2%</td>
<td>$1,637</td>
<td>$1,637 (65, $360)</td>
<td>25.2%</td>
</tr>
<tr>
<td>&gt;1,000 (n = 1)</td>
<td>0.0%</td>
<td>$2,187 (65, $360)</td>
<td>$2,187 (65, $360)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Shaw et al JACC 2009 54: 1258-1267

Statin Therapy and Plaque Regression

- Regression of Coronary Atherosclerosis by Simvastatin: A Serial Intravascular Ultrasound Study
  Jensen et al (Circulation 2004;110:265.)
  Conclusions: Lipid-lowering therapy with simvastatin for 12 months is associated with a significant plaque plus media (P+M) regression in coronary arteries measured as reduction in P+M and external elastic membrane volumes without any concomitant change in lumen volume

- Relation Between Progression and Regression of Atherosclerotic Left Main CAD and Serum Cholesterol Levels as Assessed With Serial Long-Term (>12 Months) Follow-Up Intravascular Ultrasound.
  Von Birgelen et al (Circulation 2003;108:2757-2762.)
  Conclusions: There is a positive linear relation between LDL cholesterol and annual changes in plaque size, with an LDL value of 75 mg/dL predicting, on average, no plaque progression. HDL cholesterol shows an inverse relation with annual changes in plaque size.

- Early Statin Treatment in Patients With ACS. Demonstration of the Beneficial Effect on Atherosclerotic Lesions by Serial Volumetric IVUS Analysis During Half a Year After Event: The ESTABLISH study
  Shinya Okazaki, MD (Circulation 2004;110:1061-1068.)
  Conclusions: Early aggressive lipid-lowering therapy by atorvastatin for 6 months significantly reduced plaque volume in patients with ACS. Percent change in plaque volume showed a significant positive correlation with percent LDL-C reduction, even in patients with low baseline LDL-C.

- Effects of Statin Treatments on Coronary Plaques Assessed by Volumetric Virtual Histology Intravascular Ultrasound Analysis.
  Myeong-Ki Hong (J Am Coll Cardiol Intv 2009;2:679-688)
  Conclusions: Serial volumetric IVUS showed that statin treatments might be associated with significant changes in necrotic core and fibrofatty plaque volume. The changes in both plaques’ component volume were not statistically different between simvastatin- and rosuvastatin-treated subgroups.
Change in Progression of IVUS Percent Atheroma Volume vs. LDL-C in IVUS Trials

- REVERSAL pravastatin
- CAMELOT placebo
- REVERSAL atorvastatin
- ACTIVATE placebo
- A-Plus placebo
- ASTEROID rosuvastatin

$r^2 = 0.95$  
$p < 0.001$

On-Treatment LDL-C (mg/dL)

Serial CTA for Tracking Plaque Progression
Effects of Statin Therapy

CTA in 32 PTS: 24 fluvastatin Rx; 8 controls  Serial imaging over median 12 months

Inoue et al. JACC CV Imaging 2010;3:691
**JUPITER: Cardiovascular Events**  
**The New Clinical Paradigm**

To prevent 1 primary event: treat 95 patients for 2 years or 25 patients for 5 years

**Primary End Point:** RR 0.56 (0.46-0.69), p<.00001  
**MI, Stroke, CV Death:** RR 0.53 (0.4-0.69), p<.0001

At a median of 1.9 years in a clinically low risk group, *but reclassified as high risk* by CRP, the incidence of major CV events was significantly reduced with rosuvastatin.

*CACSI (MESA-Lancet 2011)*

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**JUPITER Trial: Influence of Treatment Intensity on Patient Outcome**

17,802 pts CRP >=2mg/dl and LDL<130 mg/dl  
Median fu 2 years (1-5)

Events/1000pt-yrs.  
Placebo: 1.18  
R LDL>50mg/dl: 0.86  
R LDL<50mg/dl: 0.44

Side effects not significantly different

Hsia et al  
*JACC* 2011;57:1666-1675
Implications of CACS JUPITER Population from MESA

- 950 Patients in MESA who also met inclusion criteria for JUPITER; f/u 5.8 years
- Calculated: # of pts. treated with a statin to prevent 1 coronary or CV disease event

<table>
<thead>
<tr>
<th>CHD Events</th>
<th>CVD Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVD EVENTS:</td>
<td>CACS 0 = 124; CACS 1-100 = 54; CACS &gt;100 = 1</td>
</tr>
<tr>
<td>CHD EVENTS:</td>
<td>CACS 0 = 549; CACS 1-100 = 94; CACS &gt;100 = 2</td>
</tr>
</tbody>
</table>


Positive FH & CACS >80th Percentile Identifies Receives Benefit from Statins

St. Francis Heart Study
1,005 individuals (FH+ 543)
Mean age of 59.0±5.9 yrs.
Median CCS of 370
F/U: 4.3 yrs.

NNT to Prevent 1 Event: 18.9

Endpoints:
Cardiac Death, MI, Revascularization, Stroke, Arterial surgery

-FH: 6.6% (Active) vs. 6.8 (Placebo), p=NS

Mulders. J Am Coll Cardiol Img 2012;5:252–60
Evaluating CP In The ED : The Value of CACS?

CT CACS= 0 ;
Normal coronary arteries – the rule rather than the exception

Rapid (10-20 sec breathhold)
No Patient Preparation/contrast
Performed on standard MDCT scanners (>= 16 slice)
Relatively low radiation (1-2mSv)
Can Identify Non-coronary causes of chest pain
No contraindications

The Methodist Hospital Experience:

CACS In the ED:
Identifying Which Patients With Chest Pain Can Be Safely Discharged Home

Prospective study in 1031 Consecutive Patients with ACP, normal initial troponin, non-diagnostic ECG for ACS and no prior history of CAD admitted through the ED from September 2006 to November 2007 (all had CACS and SPECT)

20% of all ED CP Visits and 55% of those admitted with non-diagnostic CP, 99% TIMI Score <4
(99% with 6 month follow-up)

CT CACS: CP Evaluation in the ED

53 year old man: atypical chest pain, ST changes, borderline initial troponin (0.14)
ATP III Risk Score: 27% (High)

 Patients With CACS of Zero
Clinical Implications (n=625)

Overall MACE 3.1% over 7 months
CACS of 0: Very low likelihood of severe stenosis or adverse outcome

Abnormal SPECT (n=5)
- 4/5 normal LHC
- 1/5 (0.16%) treated medically without events in follow-up

CV Events (n=2)
- Both AMI (troponin I <2) on index visit but with normal gated SPECT exam
- No events during follow-up

CACS=0 : Safely Discharge home from ED, Avoid unnecessary Hospitalization and SPECT, Selectively Target Appropriate Patients For SPECT, Potential for Cost Savings!
CACS and ACS in the ED Setting

ROMICAT: ACS 1/14 (7.1%) PTS with NCP BUT only 1/197 PTS. Without CAC (0.5%)

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>N</th>
<th>CACS =0</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laudon et al</td>
<td>1999</td>
<td>105</td>
<td>59</td>
<td>59 (59%)</td>
<td>100</td>
<td>63</td>
<td>30</td>
</tr>
<tr>
<td>McLaughlin et al</td>
<td>1999</td>
<td>134</td>
<td>48</td>
<td>48 (36%)</td>
<td>100</td>
<td>38</td>
<td>8</td>
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<tr>
<td>Georgiou et al</td>
<td>2001</td>
<td>192</td>
<td>76</td>
<td>76 (40%)</td>
<td>100</td>
<td>97</td>
<td>55</td>
</tr>
<tr>
<td>Hoffman et al</td>
<td>2009</td>
<td>368</td>
<td>197</td>
<td>197 (54%)</td>
<td>97</td>
<td>59</td>
<td>18</td>
</tr>
<tr>
<td>Laudon et al</td>
<td>2010</td>
<td>263</td>
<td>133</td>
<td>133 (51%)</td>
<td>97</td>
<td>57</td>
<td>23</td>
</tr>
<tr>
<td>Nabi et al</td>
<td>2010</td>
<td>1031</td>
<td>625</td>
<td>625 (61%)</td>
<td>93.8</td>
<td>62.4</td>
<td>7.4</td>
</tr>
<tr>
<td>Fernandez-Freira</td>
<td>2011</td>
<td>225</td>
<td>133</td>
<td>133 (59%)</td>
<td>91</td>
<td>64</td>
<td>20</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td>2318</td>
<td>1271</td>
<td>1271 (55%)</td>
<td><strong>96.4</strong></td>
<td><strong>59.5</strong></td>
<td><strong>17.8</strong></td>
</tr>
</tbody>
</table>

95% CI 92-99% 57-62% 15-20% 99-100%

Screening for CAD In Asymptomatic/Symptomatic Patients: The Value of CACS

- Initial test in *asymptomatic* patients at low-intermediate clinical risk (FRS) to:
  - identify early atherosclerosis and “prevent progression”
  - detect significant *silent myocardial ischemia* and initiate therapy

- Subsequent test in *intermediate-high risk patients with a normal stress SPECT* to determine atherosclerotic burden and modify long-term risk.
- Role in the ED for evaluating low-intermediate risk patients with ACP