Interplay of Coronary Artery Calcification and Traditional Risk Factors for the Prediction of All-Cause Mortality in Asymptomatic Individuals

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Background—Current guidelines recommend the use of coronary artery calcium (CAC) scoring for intermediate-risk patients; however, the potential role of CAC among individuals who have no risk factors (RFs) is less established. We sought to examine the relationship between the presence and burden of traditional RFs and CAC for the prediction of all-cause mortality.

Methods and Results—The study cohort consisted of 44,052 consecutive asymptomatic individuals free of known coronary heart disease referred for computed tomography for the assessment of CAC. The following RFs were considered: (1) current cigarette smoking, (2) dyslipidemia, (3) diabetes mellitus, (4) hypertension, and (5) family history of coronary heart disease. Patients were followed for a mean of 5.6 ± 2.6 years for the primary end point of all-cause mortality. Among individuals who had no RF, Cox proportional model adjusted for age and sex identified that increasing CAC scores were associated with 3.00- to 13.38-fold higher mortality risk. The lowest survival rate was observed in those with no CAC and no RF, whereas those with CAC ≥ 400 and ≥ 3 RFs had the highest all-cause fatality rate. Notably, individuals with no RF and CAC ≥ 400 had a substantially higher mortality rate compared with individuals with ≥ 3 RFs in the absence of CAC (16.89 versus 2.72 per 1000 person-years).

Conclusions—By highlighting that individuals without RFs but elevated CAC have a substantially higher event rates than those who have multiple RFs but no CAC, these findings challenge the exclusive use of traditional risk assessment algorithms for guiding the intensity of primary prevention therapies. (Circ Cardiovasc Imaging. 2012; 5:467-473).

Key Words: coronary artery calcium ◼ risk factors ◼ outcome ◼ mortality

Coronary heart disease is the leading cause of death in most developed countries, including the United States. Traditionally, a risk factor (RF)—based approach has been used to identify individuals at increased risk for coronary events. For example, the Framingham Risk Score, Adult Treatment Panel III, QRISK, and other algorithms have been designed to facilitate clinical risk assessment based on the presence or absence of the traditional RFs.1-5 Despite these valuable tools, many high-risk individuals are not identified by traditional RF-based algorithms.6-10

Clinical Perspective on p 473

Measurement of coronary artery calcium (CAC) by cardiac computed tomography is a noninvasive method of quantifying the burden of coronary atherosclerosis that has been proposed as a tool to enhance traditional methods for risk stratification. Studies have demonstrated that CAC improves risk prediction beyond that of conventional RF-based algorithms,11-19 and guidelines now recommend the use of CAC for select low- to intermediate-risk individuals.20, 21 However, the frequency of high CAC burden as well its prognostic value among low-risk individuals (ie, without any known RFs) is unclear. In addition, outcomes of low-risk individuals with CAC compared with those considered to have high risk by traditional algorithms have not been reported. Therefore, in the present study we sought to examine the interplay between traditional RFs and CAC for the prediction of mortality in a large asymptomatic cohort to: (1) identify the prognostic value of CAC among...
individuals who have no reported RFs; and (2) compare the risk of individuals with no RFs who have CAC with those who have multiple RFs but no CAC.

Methods
The study cohort consisted of 44,052 consecutive asymptomatic individuals free of known coronary heart disease referred for electron beam tomography (EBT) for the assessment of subclinical atherosclerosis at 3 different centers in United States (Nashville, TN; Columbus, OH; and Torrance, CA) from 1991 to 2004. Patients were determined to be free of coronary heart disease based on patient history and prior work-up conducted by the referring physician. The combined population was predominantly white and middle-aged.

Study participants were referred by their primary physicians for the assessments of subclinical atherosclerosis and, as such, do not represent a random sample of the general population. All screened individuals provided informed consent to undergo EBT and for the use of their blinded data for epidemiological research. The general study received approval from the Human Investigations Committee, and separate Committee approval was obtained for the patient interviews, collection of baseline and follow-up data, and corroboration of the occurrence of death. The methods have been previously described in detail.21

RF Data Collection
All study participants were given a questionnaire for the collection of demographic and clinical characteristics as well as baseline cardiovascular RFs. The following RFs were considered: (1) current cigarette smoking was considered present if a subject was a smoker at the time of scanning; (2) dyslipidemia was considered to be present for any individual reporting a history of high total cholesterol, high low-density lipoprotein-cholesterol, low high-density lipoprotein-cholesterol, and high triglycerides, or current use of lipid-lowering therapy; (3) diabetes mellitus was defined as the use of oral anti–diabetes mellitus medications or insulin; (4) hypertension was defined as a self-reported history of high blood pressure or use of antihypertensive medication; and (5) family history of premature coronary heart disease (CHD) was determined by asking patients whether any member of their immediate family (parents or siblings) had a history of fatal or nonfatal myocardial infarction and coronary revascularization. In 36,010 (82% of the study population) study participants, a premature history of CHD was defined if such events occurred before 55 years of age in male relatives or before 65 years of age in female relatives. In 8,042 (18% of the study population) study participants, a premature history of CHD was defined if such events occurred before 55 years of age in both male and female relatives.6

EBT Screening Protocol
All subjects underwent EBT on either a C-100 or a C-150 Ultrafast computed tomography scanner (GE-Imatron, South San Francisco, CA). Using a tomographic slice thickness of 3 mm, a total of ≥40 sections were obtained beginning at the level of the carina and proceeding caudally to the level of the diaphragm. Images were obtained using a 100 ms/slice scanning time, with image acquisition electrocardiographically triggered at 60% to 80% of the R-R interval. A calcified lesion was defined as ≥2 contiguous pixels, with a peak attenuation of at least 130 Hounsfield units. Each lesion was then scored using the method developed by Agatston et al.21

Follow-Up and Mortality Ascertainment
Patients were followed for a mean of 5.6±2.6 years (median, 5 years; range, 1–13 years). Ascertainment of mortality was conducted by individuals blinded to baseline historical data and EBT results. The occurrence of death was verified using the Social Security Death Index. A full Social Security Death Index search was successfully completed in 100% of patients.

Statistical Methods
The baseline characteristics of the study population are presented by the specified CAC group (0, 1–100, 101–400, and ≥400) and in aggregate for the entire study population. Age is presented as a continuous measure±SD, and other risk variables are expressed as proportional frequencies. Age was compared across increasing CAC groups using ANOVA techniques, and proportional frequencies of other risk variables were compared across increasing CAC groups using χ² analysis. P<0.05 was considered statistically significant.

Annualized all-cause mortality rates were estimated by dividing the number of deaths by the number of person-years at risk. In addition, survival analysis was conducted using individual subject time-to-all-cause mortality data. Curves representing the cumulative probability of survival were generated using Kaplan-Meier estimates stratified by categories of increasing CAC as well as by increasing the number of RFs. The logrank test was used to compare for differences in survival between subgroups. To evaluate the effect of CAC or RF burden on all-cause mortality, hazard ratios and 95% CIs were calculated using the Cox proportional hazards regression model, adjusted for age and sex.

We computed receiver operating characteristic curves and tested for equality of the areas under the curves to examine whether CAC added incremental predictive value over a baseline model that included age, sex, and RFs. In a similar fashion, we also assessed whether increasing number of RFs and age/sex would add predictive incremental predictive value once CAC was taken into account. In addition, to further evaluate the potential incremental value of using CAC over RFs and age/sex, the net reclassification improvement was calculated by the method previously described by Pencina et al.24 Because an underlying assumption of this statistic is that individuals in different risk groups are managed differently, the following risk categories were used: (1) RF model—low risk: no RF; intermediate risk: 1 to 2 RFs; high risk: ≥2 RFs; (2) CAC model—low risk: CAC=0; intermediate risk: CAC 1 to 100; high risk: CAC>100. When applied to our analysis, the net reclassification improvement thus estimates the extent to which people who died were appropriately reclassified up (or inappropriately classified down) as well those who survived were appropriately reclassified down (or inappropriately classified up). All statistical analyses were performed using Stata version 10 (STATA Corp, College Station, TX).

Results
The clinical characteristics of the 44,052 subjects are shown in Table 1. The mean age of the study population was 54±10 years, and 54% were men. More than one third (43%; n=18,819) of the subjects reported no RF. A total of 19,898 patients (45%) had no CAC on screening EBT, whereas 14,181 (32%) had CAC scores of 1 to 100, 5,739 (13%) had CAC scores of 101 to 400, and 4,234 (10%) had CAC>400. As shown in Figure 1, more than half of individuals with no RF had CAC=0 (53%), whereas those with RFs were less likely to demonstrate CAC=0. CAC scores of 1 to 100, 101 to 400, and >400 were seen in 32%, 10% and 6% individuals with no RF. In comparison, the respective prevalence was 32%, 19%, and 17% among those with ≥3 RFs.

Overall, there were 901 deaths (2.05%) in the total study population over a mean follow-up of 5.6±2.6 years (median, 5.0 years; range, 1–13 years). The annualized mortality rate was 1.84 deaths per 1000 person-years (95% CI, 1.62–2.09) for those with no RF compared with 4.13 (95% CI, 3.60–4.75), 5.78 (95% CI, 5.07–6.59), and 9.11 (95% CI, 8.00–10.38) deaths per 1000 person-years among those with 1, 2, and ≥3 RFs, respectively. On the other hand, the annualized mortality rate was 0.87 deaths per 1000 person-years (95% CI, 0.72–1.06) for those with CAC=0 compared with 2.97 (95% CI,
Using Cox proportional hazards regression, after adjusting for age and sex, increasing CAC scores were more strongly associated with all-cause mortality among those with no RF compared with individuals with RF as shown in Table 3. Figure 2 shows reduced survival curves with increasing CAC score at each level of baseline RF burden. Figure 3 shows that the lowest event rate was observed in those with no CAC and no RF, whereas those with CAC $\geq 400$ and $\geq 3$ RFs had the highest all-cause mortality rate. Of note, individuals with no RF and CAC $\geq 400$ had a much higher mortality rate compared with individuals with $\geq 3$ RFs but absent CAC (16.89 per 1000 person-years versus 2.72 per 1000 person-years). Similar trend was noted when in addition age (men $\geq 45$ years and women $\geq 45$ years) was also considered as an RF (online-only Data Supplement).

Among those with CAC=0 at baseline ($n=19,895$), the respective median 5-year all-cause survival was 99.7%, 99.3%, 99.3%, and 99.0% with the presence of 0, 1, 2, and $\geq 3$ RFs respectively. In Cox regression analyses adjusted for age and sex, increasing RFs were associated with a higher hazard ratio of all-cause mortality across all CAC score categories (Table 4).

When predicting all-cause mortality, the addition of CAC to a model containing age, sex, and RFs resulted in a significant incremental improvement with the receiver operating characteristic curve increasing from 0.76 (95% CI, 0.75–0.78) to 0.81 (95% CI, 0.79–0.82). Finally, in comparison with a model based on the number of traditional RFs present, the model using CAC resulted in a net reclassification improvement of 0.36 ($P<0.001$) (online-only Data Supplement).

**Discussion**

In this large cohort of 44,052 asymptomatic subjects followed for a median of 5 years, we demonstrate that individuals without RFs but elevated CAC (who are generally not candidate for aggressive prevention) had a significantly higher mortality rate compared with those with RFs but absent CAC (16.89 per 1000 person-years versus 2.72 per 1000 person-years). Similar trend was noted when in addition age (men $\geq 45$ years and women $\geq 45$ years) was also considered as an RF (online-only Data Supplement).
Figure 2. Kaplan-Meier survival curves by coronary artery calcium (CAC) scores across increasing risk factor (RF) burden.

Figure 3. Mortality rate (per 1000 person-years) with increasing coronary artery calcium (CAC) scores according to burden of risk factors (RFs).

<table>
<thead>
<tr>
<th>CAC Score</th>
<th>0 RF</th>
<th>1 RF</th>
<th>2 RF</th>
<th>≥3 RF</th>
<th>Total</th>
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<tr>
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<td>9,805</td>
<td>4,558</td>
<td>3,322</td>
<td>2,123</td>
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<tr>
<td>CAC 1-100</td>
<td>5,994</td>
<td>3,250</td>
<td>2,913</td>
<td>2,204</td>
<td>14,181</td>
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<td>CAC 101-400</td>
<td>1,883</td>
<td>1,301</td>
<td>1,371</td>
<td>1,184</td>
<td>5,739</td>
</tr>
<tr>
<td>CAC&gt;400</td>
<td>1,047</td>
<td>984</td>
<td>1,148</td>
<td>1,055</td>
<td>4,234</td>
</tr>
<tr>
<td>Total</td>
<td>18,819</td>
<td>10,093</td>
<td>8,754</td>
<td>6,386</td>
<td>44,052</td>
</tr>
</tbody>
</table>
higher mortality rates than individuals with multiple RFs but no CAC. These findings challenge the exclusive use of traditional risk assessment algorithms for determining the intensity of primary prevention therapies and suggest that selected groups of patients without RFs may benefit from further risk assessment and preventive therapies.

Value of CAC Testing in Low-Risk Individuals

Previous studies have examined the prevalence and significance of subclinical atherosclerosis in individuals traditionally considered to be at low risk for events. Michos et al\(^2\) showed that 84% of women with significant CAC were classified as low risk for cardiovascular events, and Greenland et al\(^3\) found that in a group of subjects with few or no RF, 53% had detectable CAC and 19% had CAC>300. In another study, Lakoski et al\(^4\) demonstrated that 32% of women classified as low risk have detectable CAC and 4% have a CAC≥300. They were also able to show that among low-risk women, those with CAC were at increased risk for cardiovascular events compared with those without CAC. The findings from the study are consistent with these reports: 43% of the subjects in our cohort had no RF, and within this subgroup, 48% had detectable CAC and 6% had a CAC>400.

Other studies have shown similar results: Shaw et al\(^5\) demonstrated that the 5-year mortality rate for patients with few or no RF was 0.9% among subjects with a CAC score <10 but increased to 3.9% among individuals with a CAC score≥1000. They also demonstrated that subjects at low risk by traditional risk assessment models with a CAC≥1000 had a higher 5-year mortality rate (3.9%) than subjects at high risk with a CAC<10 (2.8%).\(^6\) However, Greenland et al\(^7\) did not observe an increased event rate in the low-risk, high CAC group, probably because of a small sample size (for the low-risk group, n=98). Despite this, subjects at intermediate risk with CAC≥300 were at greater risk for coronary events or nonfatal myocardial infarction than subjects at high risk without CAC.

Our study adds to current literature by evaluating for the first time the value of subclinical atherosclerosis screening for the first time in the largest cohort of individuals with no RF and demonstrating an incremental increase in risk of all-cause mortality with increasing CAC scores. These findings have important implications for guidelines regarding identifying appropriate candidates for CAC testing. Although prior guidelines only recommended CAC testing for select intermediate-risk individuals with 10-year risk of 10% to 20%,\(^8,9\) updated guidelines have now acknowledged the CAC testing for further risk stratification in lower risk individuals and had reduced the threshold to include individuals with estimated 6% to 10% risk of CHD in next 10 years.\(^10\) However, current guidelines recommend against CAC testing in those with 0 to 1 RF. Our study findings suggest that CAC testing even among those with no RF provides important prognostic information, which can be instrumental in guiding preventive therapies. Nevertheless, our findings need to be validated in additional settings before any changes in the guidelines for CAC testing are adopted. Current efforts are underway to test this hypothesis in large prospective studies as well as to evaluate whether this approach can potentially be cost-effective. In the context of potentially expanding the eligibility criteria for population to be considered candidate for screening, issues such as identifications of incidentalomas require thoughtful consideration, which may result in potential unnecessary downstream testing and healthcare costs.

Power of 0 in Traditional High-Risk Individuals

Although high CAC scores can be useful in identifying high-risk individuals among those with no RF, equally important is the fact that the absence of CAC confers a low risk for future CVD events and mortality across all range of RF burden. Sarwar et al\(^11\) in a meta-analysis showed that among 29,312 individuals without evidence of CAC, only 0.56% of subjects without CAC experienced a cardiovascular event during a mean follow-up period of 51 months. These findings were confirmed in a large retrospective study\(^12\) and a multiethnic prospective study,\(^13\) demonstrating a low event risk with the absence of CAC in asymptomatic individuals. A recent study by Malik et al\(^14\) also recently demonstrated that more than one third of individuals with diabetes mellitus (38%) have no detectable CAC and minimal CVD events in ≥6 years of follow-up. Blaha et al\(^15\) also showed that nearly half of the individuals meeting eligibility for statin therapy based on Justification for the Use of Statins in Prevention: an Intervention Trial Evaluating Rosuvastatin (JUPITER) criteria had no CAC and experienced an extremely low event rate, with an unfavorable estimated number needed to treat for 5 years of 549 to prevent 1 CHD event compared with 42 among those with the presence of CAC.

In this regard, the absence of CAC can be used to identify individuals with an extremely low risk in whom lifestyle interventions may be advocated while safely deferring the use of costly pharmacotherapy.\(^16\) Such a strategy could enable us to focus on individuals with actual disease (as opposed to ones who have RFs but may never develop any substantial coronary atherosclerosis) as they are the ones in whom the majority of clinical events will occur and are most likely to benefit from more aggressive preventive therapies.
Limitations
There are a few limitations to this study. First, all patients were referred for CAC screening and, therefore, do not represent a random sample of the population. In general, patients referred for CAC scans may be at higher risk compared with age-matched patients from the general population. If this were the case in the present study, the finding of excellent survival among patients with 0 CAC could be considered even more striking. A second potential weakness is the self-reporting of RFs. Data collected by self-report are limited by patient recall and thus subject to recall bias. Although Hoff et al have shown a good reliability of self-reported histories of CHD RFs in self-referred individuals for EBT scanning, because the CHD RFs were self-reported, the potential residual confounding cannot be ruled out, thus possibly diminishing the strength of association of RFs with mortality. Nevertheless, in spite of the fact that RFs may be under-reported, the absence of CAC was still associated with favorable prognosis across all levels of RFs. Because of the aforementioned limitations, our findings need to be verified in cohorts with well-measured RFs.

Our models do not include the cause of death, and, as such, some mortality events may not be related to atherosclerotic disease. However, all-cause mortality is an appropriate end point to follow, because when one accounts for both cardiac and systemic forms of the disease, nearly three-fourths of all deaths have been related to atherosclerosis. Furthermore, this end point is unaffected by reporting and misclassification bias potentially introduced by death reports.

In addition, more detailed conclusions in our study are not possible because of the lack of cardiovascular-specific mortality data. Although coronary heart disease remains the most common killer in industrialized countries, it is not possible to ascertain the proportion of deaths that are cardiovascular in origin. Although CAC is presumed to influence mortality mainly via cardiovascular mechanisms, other RFs such as smoking, hypertension, and diabetes mellitus contribute to all-cause mortality via additional noncardiovascular mechanisms (ie, lung disease and kidney dysfunction). Finally, the net reclassification improvement with CAC reported in our study is slightly higher than those seen in other prospective studies and can be because of the fact that RFs were not directly measured.

Conclusions
Our study findings support a paradigm shift in CVD risk assessment from RF-based approach to detection of subclinical atherosclerosis burden as evident by the fact that a significant proportion of those with no RF have a severe amount of coronary atherosclerosis and have a high risk for all-cause mortality. The higher precision of CAC relative to RFs for identifying at-risk individuals may be because of the fact that CAC is a measure of actual disease that occurs further down the causal pathway than the presence of RFs that are mere surrogates for this process. Whether this paradigm shift will eventually result in more appropriate allocation of resources and reduce the overall economic healthcare costs is yet to be answered and needs to be addressed in future randomized trials.

Disclosures
Dr Budoff is on the Speaker Bureau of General Electrics (GE). The other authors have no conflicts to report.

References


**CLINICAL PERSPECTIVE**

Current guidelines recommend the use of coronary artery calcium (CAC) scoring for select intermediate-risk patients. In the present study, we followed 44,052 consecutive asymptomatic individuals for a median of 5 years. Subjects without risk factors (obtained from patients) but elevated CAC had a significantly higher event rate than individuals with multiple risk factors but no CAC. Conversely, the absence of CAC was associated with a favorable prognosis even among those with multiple risk factors. These findings suggest a potential benefit of CAC over traditional risk assessment to tailor primary prevention therapy. However, further studies based on prospectively collected and quantitative data on risk factors are needed to determine whether CAC in the absence of risk factors enable more effective treatment and reduce overall healthcare costs.
Interplay of Coronary Artery Calcification and Traditional Risk Factors for the Prediction of All-Cause Mortality in Asymptomatic Individuals

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Supplemental Figures and Figure Legends

Mortality Rate (per 1000 person-years) With Increasing Coronary Artery Calcium Scores according to Burden of Risk Factors (with addition of age as risk factor, men ≥45 years and women ≥45 years)

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<th></th>
<th>0 RF</th>
<th>1 RF</th>
<th>2 RF</th>
<th>≥3 RF</th>
<th>Total</th>
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<td>15,490</td>
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### Supplemental Table

**Individuals with events**

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<th>Intermediate (CAC 1-100)</th>
<th>High (CAC&gt;100)</th>
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**Individuals with no events**

<table>
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<th>Intermediate (CAC 1-100)</th>
<th>High (CAC&gt;100)</th>
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<td>RF≥3</td>
<td>2,101</td>
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Reclassification tables for individuals with and without mortality are used to demonstrate the number of individuals who were reclassified appropriately (green) versus inappropriately (red).