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Arterial Age as a Function of Coronary Artery Calcium (From the Multi-Ethnic Study of Atherosclerosis [MESA])

Robyn L. McClelland, PhD^a, Khurram Nasir, MD MPH^b, Matthew Budoff, MD^c, Roger S. Blumenthal, MD^d, and Richard A. Kronmal, PhD^a

a Department of Biostatistics, University of Washington

b Massachusetts General Hospital

c Division of Cardiology, Harbor-UCLA Medical Center

d Ciccarone Preventive Cardiology Center, Johns Hopkins University

Abstract

It has been proposed that coronary artery calcium (CAC) can be used to estimate an arterial age in adults. Supporting this concept is that chronologic age, as used in cardiovascular risk assessment, is a surrogate for atherosclerotic burden. This measure can provide the patient with a more understandable version of their CAC score (e.g. you are 55 years old, but your arteries are more consistent with an arterial age of 65). We describe a method of estimating arterial age by equating estimated coronary heart disease (CHD) risk for observed age and coronary artery calcium (CAC). Arterial age is then the risk-equivalent of coronary artery calcium. We use data from the Multi-Ethnic Study of Atherosclerosis (MESA), a cohort study of 6814 participants free of clinical cardiovascular disease, followed for an average of 4 years. Estimated arterial age is obtained as a simple linear function of log-transformed CAC. In a model for incident CHD risk controlling for both age and arterial age, only arterial age was significant, indicating that observed age does not provide additional information after controlling for arterial age. Framingham risk calculated using this arterial age is more predictive of short-term incident coronary events than Framingham risk based on observed age (area under the ROC curve 0.75 for Framingham risk based on observed age, 0.79 using arterial age, $p=0.006$). In conclusion, arterial age provides a convenient transformation of CAC from Agatston units to a scale more easily appreciated by both patients and treating physicians.

Introduction

The concept of vascular or arterial age has been proposed in several previous studies. The idea is to adjust the age of a patient based on their level of atherosclerosis. Stein et al [1] have developed an algorithm for calculating vascular age based on carotid IMT. Others have proposed algorithms based on coronary artery calcium [2–5]. The primary motivation for these calculations is to provide the patient with a more easily understandable version of their atherosclerosis burden (e.g. you are 55 years old, but your arteries are more consistent with a vascular age of 65 years). Additionally, it has been suggested that such a biologic age be

Corresponding Author: Robyn McClelland, PhD, Collaborative Health Studies Coordinating Center, Department of Biostatistics, University of Washington, Building 29, Suite 310, 6200 NE 74th St, Seattle, WA, 98115, Telephone: 206-897-1956, Fax: 206-616-4075, Email: rmcclell@u.washington.edu.

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substituted for observed age in calculation of the Framingham Risk Score, an idea first proposed by Grundy [6,7]. This would allow clinicians to continue to use existing scoring algorithms, but have a more refined and accurate estimation of future cardiovascular risk. Although a few methods have been proposed, the issue of how to best estimate the arterial age remains an open question. We propose estimating arterial age by equating estimated CHD risk for observed age and (log-transformed) CAC, using data from the Multi-Ethnic Study of Atherosclerosis (MESA). That is, the arterial age for a participant is the age at which the estimated CHD risk (modeled as a function of age) is the same as that for the observed CAC score. Arterial age is then the risk-equivalent of coronary artery calcium. In essence, this is a simple transformation of observed CAC from Agatston units to “age units”. We show that chronologic or observed age does not provide any additional information over this estimated arterial age. Additionally we provide tables and figures allowing easy translation of an observed CAC score to arterial age, including 95% confidence limits. Finally, we show that Framingham risk calculated using this arterial age is more predictive of incident events than Framingham risk based on observed age.

Methods

MESA is a prospective cohort study of the prevalence, risk factors, and progression of subclinical cardiovascular disease in a multi-ethnic community-based cohort. A detailed description of the study design and methods has been published previously [8]. Briefly, 6814 participants aged 45–84 years who identified themselves as White, African-American, Hispanic, or Chinese were recruited from 6 U.S. communities from 2000–2002. All participants were free of clinically apparent cardiovascular disease at study entry. The communities were Forsyth County, NC; Northern Manhattan and the Bronx, NY; Baltimore City and Baltimore County, MD; St. Paul, MN; Chicago, IL; and Los Angeles County, CA.

Each field center developed its sampling frame according to the characteristics of its community and available resources, including lists of residents, dwellings, telephone exchanges, Division of Motor Vehicle lists, consumer lists, voter registration lists, and census data. Selection from the sampling frames used either simple random samples from the lists, random-digit dialing, or proceeded sequentially through pre-defined neighborhoods. Each site recruited an approximately equal number of men and women, according to pre-specified age and race/ethnicity proportions. All participants gave informed consent.

Coronary artery calcium was measured using either electron-beam computed tomography at 3 field centers, or multi-detector computed tomography at 3 field centers. Each participant was scanned twice consecutively and these scans were read independently at a centralized reading center. Measurements were made on all participants baseline (2000–2002) and at 1 of 2 follow-up examinations in either 2002–2003 or 2003–2005. The methodology for acquisition and interpretation of the scans has been documented previously [9]. The results from the 2 scans were averaged in order to provide a more accurate point estimate of the amount of calcium present. The amount of calcium was quantified using the Agatston scoring method [10]. Calcium scores were adjusted using a standard calcium phantom that was scanned along with the participant [11]. The phantom contained 4 bars of known calcium density. This phantom provides a way of calibrating the degree of brightness between sites and participants.

The current report includes an average of 4 years of follow-up. At intervals of 9–12 months, a telephone interviewer contacted each participant to inquire about interim hospital admissions, cardiovascular outpatient diagnoses, and deaths. To verify self-reported diagnoses, we requested copies of all death certificates and medical records for hospitalizations and outpatient cardiovascular diagnoses and conducted next-of-kin interviews for out of hospital cardiovascular deaths. Trained personnel abstracted medical records suggesting possible

cardiovascular events. Two physicians independently classified and assigned incidence dates. If, after review and adjudication, disagreements persisted, a full mortality and morbidity review committee made the final classification.

For purposes of this study, we used all incident CHD events as the endpoint, including definite or probable myocardial infarction, resuscitated cardiac arrest, fatal CHD, definite angina, and probable angina if accompanied by revascularization. Definitions for each of these events are as follows. Reviewers classified myocardial infarction as definite, probable, or absent based primarily on combinations of symptoms, electrocardiogram, and cardiac biomarker levels. In most cases, definite or probable myocardial infarction required either abnormal cardiac biomarkers (2 times upper limits of normal) regardless of pain or electrocardiogram findings; evolving Q waves regardless of pain or biomarker findings; or a combination of chest pain, and ST-T evolution or new left bundle branch block, and biomarker levels 1–2 times upper limits of normal. Reviewers classified resuscitated cardiac arrest when a patient successfully recovered from a full cardiac arrest through cardiopulmonary resuscitation (including cardioversion). Angina was classified, except in the setting of myocardial infarction, as definite, probable, or absent. Definite or probable angina required symptoms of typical chest pain or atypical symptoms, as asymptomatic coronary artery disease is not a MESA endpoint. Probable angina required, in addition to symptoms, a physician diagnosis of angina and medical treatment for it. Definite angina required one or more additional criteria, including coronary artery bypass graft surgery or other revascularization procedure; $\geq 70\%$ obstruction on coronary angiography; or evidence of ischemia by stress tests or by resting electrocardiogram. Fatal CHD required a documented myocardial infarction within the previous 28 days, chest pain within the 72 hours before death, or a history of CHD, and required the absence of a known non-atherosclerotic or non-cardiac cause of death.

We model the risk of incident CHD (all CHD including angina) first as a function of age, then as a function of $\log(\text{CAC}+1)$ using an exponential survival model (log refers to the natural logarithm). Specifically, the conditional failure rate or hazards function was modeled as:

$$h(t|\text{age}) = \exp(\beta_0 + \beta_1 \text{age})$$

and

$$h(t|\text{cac}) = \exp(\gamma_0 + \gamma_1 \log(\text{cac}+1)).$$

Equating the hazards and solving for age yields the following estimate of arterial age, plugging in the maximum likelihood estimates for each parameter:

$$\widehat{A} = \frac{(\widehat{\gamma}_0 - \widehat{\beta}_0) + \widehat{\gamma}_1 \log(\text{cac}+1)}{\widehat{\beta}_1}.$$

We estimate the variance of arterial age via the delta method as,

$$\text{var}(\widehat{A}) \approx \left(\frac{\partial \widehat{A}}{\partial \widehat{\theta}} \right) \Sigma \left(\frac{\partial \widehat{A}}{\partial \widehat{\theta}} \right)^T$$

where $\left(\frac{\partial \widehat{A}}{\partial \widehat{\theta}} \right)$ is the row vector of partial derivatives of A with respect to each parameter in $\widehat{\theta} = (\widehat{\beta}_0, \widehat{\beta}_1, \widehat{\gamma}_0, \widehat{\gamma}_1)$ and Σ is the variance covariance matrix of $\widehat{\theta}$. Robust standard errors are used. The covariance between $(\widehat{\beta}_0, \widehat{\beta}_1)$ and $(\widehat{\gamma}_0, \widehat{\gamma}_1)$ is obtained via seemingly unrelated estimation [12]. The partial derivatives necessary for the above calculations are,

$$\left(\frac{\partial \widehat{A}}{\partial \theta}\right) = \left(\frac{\partial \widehat{A}}{\partial \beta_0}, \frac{\partial \widehat{A}}{\partial \beta_1}, \frac{\partial \widehat{A}}{\partial \gamma_0}, \frac{\partial \widehat{A}}{\partial \gamma_1}\right) = \left(-\frac{1}{\beta_1}, -\frac{1}{\beta_1^2}(\widehat{\gamma}_0 - \widehat{\beta}_0 + \widehat{\gamma}_1 \log(cac+1)), \frac{1}{\beta_1}, \frac{1}{\beta_1} \log(cac+1)\right).$$

A Cox proportional hazards model was fit including both vascular age and observed age to determine if observed age contributed any additional information conditional on vascular age. Separate Cox models were fit using either of vascular age or observed age, and controlling for all the Framingham risk factors including gender, total cholesterol, HDL-cholesterol, systolic and diastolic blood pressures, antihypertensive medication use, and cigarette smoking status (never/former/current), and excluding diabetics. Predicted values from these models were used to construct receiver-operator characteristic (ROC) curves for incident CHD. The area under the ROC curve is a measure of the ability to discriminate between those who experience a CHD event, and those who do not. A perfect predictor would have an area under the curve of 1.0, while a coin toss has an area of 0.5. Additionally we compared the Framingham estimated 10 year risk calculated using observed age, and using estimated arterial age. For this we used the point-based scoring algorithm as described in the National Cholesterol Education Program (NCEP) Adult Treatment Panel III report [13]. When using estimated arterial age, the points assigned for risk factors in an age-specific manner (smoking and cholesterol) used observed age, arterial age was used only to calculate age points.

Results

The MESA cohort is comprised of 53% women, with 39% of the cohort Caucasian, 12% Chinese American, 28% African American, and 22% Hispanic American. The average age is 62 years, with a range from 45–84 years. Over an average of 4 years of follow-up, 189 incident CHD events have been observed in MESA, including 108 hard CHD events (myocardial infarction and CHD death). Estimated arterial age is given by,

$$\widehat{A} = 39.1 + 7.25 \log(cac+1)$$

with approximate standard error given by,

$$se(\widehat{A}) \cong \sqrt{13.7 - 6.5 \log(cac+1) + 0.8(\log(cac+1))^2}.$$

The relationship between estimated arterial age and $\log(CAC+1)$ is displayed in Figure 1. For ease of practical use, we have labeled the x-axis using untransformed CAC values, though the plot is versus log-transformed CAC. This figure can be used to look up a particular CAC score and get an approximate arterial age and corresponding 95% confidence intervals.

In Figure 2, we illustrate the difference between arterial age and observed age, across increasing CAC values. As expected, for large CAC values arterial age tends to be higher than observed age, and for low values of CAC arterial age is lower than observed age. For CAC at the extremes, arterial age may be substantially higher or lower than observed age, sometimes as much as 40 years. This can occur for example in an older person who has zero CAC, or conversely, in a young person with substantial CAC. Table 1 provides the estimated arterial age with 95% confidence interval for various CAC scores.

In a model for incident CHD risk controlling for both age and arterial age, only arterial age was significant ($p < 0.001$ for arterial age, $p = 0.20$ for observed age). Interestingly, in a model adjusting for the Framingham risk factors and either age or arterial age, the coefficients for age and arterial age were quite similar (0.050 and 0.054 per year respectively). Despite this, the predicted risk associated with the vascular age model was a much stronger predictor of incident

CHD than the predictions based on observed age (area under the ROC curve 0.83 versus 0.76, $p < 0.0001$).

As was originally proposed by Grundy [6,7], we substituted arterial age directly in place of observed age in the existing Framingham scoring algorithms. Using the point score system described in the NCEP Adult Treatment Panel III report [13] we calculated 2 versions of Framingham risk, one using observed age and one using vascular age in place of observed age. Diabetics were excluded from these calculations as diabetes is considered a CHD risk equivalent. Figure 3 displays the difference between the 2 risk estimates as a function of log transformed CAC. Since Framingham risk is an increasing function of age, the pattern mimics that seen for the difference between observed and arterial age. That is, for high values of CAC, the estimated Framingham risk is often much higher using arterial age in place of observed age. For low values of CAC, the estimated Framingham risk may be much lower than what would be estimated based on observed age.

In terms of risk categories (<10%, 10–20% and >20% estimated 10-year CHD risk), 28% of participants would be reclassified into a different risk set using arterial age in place of observed age (16% would go to a lower risk stratum, and 12% to a higher risk stratum). Figure 4 displays the receiver-operator characteristic curves for these measures in terms of predicting incident CHD events. The score based on arterial age performs significantly better (area under the ROC curve 0.75 for Framingham risk based on observed age, 0.79 using arterial age, $p = 0.006$). The optimal area under the ROC curve for our data (obtained by fitting a model with arterial age and all the Framingham risk factors, rather than using the scoring system) was 0.82.

At the MESA public website <http://www.mesa-nhlbi.org> (under the “CAC Tools” section) we have created an online arterial age tool, where one can enter an observed CAC score and obtain the corresponding estimated arterial age. Additionally, one has the option of entering the Framingham risk factors and obtaining the estimated 10-year Framingham hard CHD risk using both chronologic age and arterial age.

Discussion

Most previous studies have estimated arterial age as the age for which the expected CAC is equal to the observed CAC. In Shaw et al [3], observed age is regressed on observed CAC, and the “biologic age” is then estimated by the fitted values from this model. A feature of this model is that anyone with a zero CAC will be assigned the model intercept, which in our data was just slightly below the average age of the cohort. Thus, anyone below the average age of the cohort with a zero score will have a higher vascular age than observed age, despite having no calcium. In our population-based cohort half of the participants have zero CAC, and of those two-thirds are under the average age of the cohort, so this method was not sensible for our data.

Other methods make use of age, gender, and/or race specific percentiles and estimate arterial age as the age at which median CAC equals observed CAC [2,4,5]. Relative ranking within a subgroup places subjects with similar CAC at vastly different risks. Gender specific ranking for instance, puts a woman with a given CAC at a higher percentile than a man, because CAC is less common in women than men. Analogously, gender-specific arterial age would be higher for the woman than the man, implying that a given CAC carries a worse prognosis in women than in men. Analysis of CAC and events in MESA suggests that this is not the case, and that although a given level of CAC is less likely to be seen in the woman, it has the same impact on risk when it is. Of course, gender will need to factor into evaluation of a patient’s overall risk (just like any other risk factor such as hypertension, diabetes, cholesterol, etc). Despite the fact that our arterial age itself is not gender (or race) specific, we note that 2 participants with the same arterial age will have (via the Framingham equations) different absolute risk estimates

depending on their gender and other risk factors. That is, the age points are assigned and scored differently by gender, and hence even though arterial age is not gender-specific, absolute risk remains higher for men than women.

In contrast to previous approaches, our method assigns an arterial age that has the same expected CHD risk as the observed CAC. We feel this approach has several advantages. Most importantly, it captures the biologic relationship that we feel is implied by the concept of an arterial age, namely that age is essentially a surrogate marker for atherosclerotic burden in terms of CHD risk prediction. For a particular CAC score, the corresponding arterial age should then be the chronologic age that conveys the same risk as that CAC score, and not necessarily the age at which an average person would be expected to have that much CAC. Additionally, the equation for converting CAC to age is simple, and approximate 95% confidence intervals are also available, to provide some indicator of the uncertainty in the transformation. In terms of communicating test results to patients, the arterial age may be a useful tool. In a prior study conducted by Goldman et al [14] they found that a strategy translating cholesterol results into risk-adjusted age was preferred by study participants, who found it easier to grasp than the absolute results or visual representations of 10-year Framingham risk. In a randomized controlled trial Parkes et al [15] found that a strategy of telling smokers their “lung-age” significantly improved the likelihood of them quitting smoking.

There are some limitations to our study that should be noted. Our follow-up is currently limited to an average of 4 years, and so the risk-equivalency of arterial age with CAC is in terms of short-term risk. As longer follow-up becomes available the relationship can be updated and evaluated to see if the relationship between CAC and arterial age is different for intermediate or long term risk. As the relationship between CAC and other endpoints, such as stroke or congestive heart failure, is different from its association with CHD, arterial age does not generalize to these other endpoints. For instance, CAC is more weakly associated with stroke than with CHD; hence, a high CAC would not translate into as high an arterial age in terms of stroke risk as for CHD risk. Additionally, validation in other cohorts is needed to determine whether the arterial age equations are robust across different populations. Finally, we do not address in this paper whether one should measure CAC. There are costs, risks and benefits to any diagnostic test and we have not attempted to weigh these tradeoffs. Rather, CAC is currently often measured in practice, and our paper suggests one way to make use of this information given that it has already been collected.

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Figure 1. Estimated Arterial Age and 95% Confidence Bands by Coronary Artery Calcium

The estimated arterial age with 95% confidence bands is displayed for each CAC score. CAC is displayed on the log scale, but labeled in original units for ease of use. A person with a CAC score of 10 Agatston units has an estimated arterial age of approximately 56, while a CAC score of 400 yields an arterial age of 83.

Figure 2. Arterial Age Minus Observed Age by Coronary Artery Calcium

For each participant this plot displays the difference between their estimated arterial age and their observed chronologic age. A negative difference indicates that their arterial age is less than their chronologic age (e.g. older participants who are free of CAC). A positive difference indicates that their arterial age is greater than their chronologic age. CAC is displayed on the log scale, but labeled in original units for ease of use.

Figure 3. Estimated Framingham Risk Using Chronologic Age Minus Estimated Risk Using Arterial Age Versus Coronary Artery Calcium

For each participant this plot displays the difference between their 10-year Framingham risk calculated using their arterial age and that calculated using their chronologic age. A negative difference indicates that their 10-year CHD risk based on arterial age is less than what would be estimated given their chronologic age (e.g. older participants who are free of CAC). A positive difference indicates that their estimated 10-year risk using arterial age is greater than that using their chronologic age. CAC is displayed on the log scale, but labeled in original units for ease of use.

Figure 4. Receiver-Operator Characteristic (ROC) Curve for Incident CHD Using Framingham Risk Based on Chronologic Age and Based on Arterial Age

Each curve indicates the ability of the predictor to discriminate between incident CHD cases and non-cases. Better discrimination is indicated by curves which are closer to the upper left hand corner, where both sensitivity and specificity are high. Random assignment, as in a coin toss, would fall along the straight line. The curve for Framingham risk score calculated using arterial age rather than chronologic age is higher, indicating better discrimination between CHD cases and non-cases.

Table 1

Estimated Arterial Age and 95% Confidence Intervals by Coronary Artery Calcium Score

CAC	Arterial Age in Years (95% CI)	CAC	Arterial Age in Years (95% CI)
0	39 (32–46)	100	73 (71–74)
10	56 (53–60)	200	78 (75–80)
20	61 (59–63)	300	80 (78–83)
30	64 (62–66)	400	83 (79–86)
40	66 (65–67)	500	84 (80–88)
50	68 (67–69)	750	87 (83–92)
60	69 (68–70)	1000	89 (84–94)
70	70 (69–71)	1500	92 (87–98)
80	71 (70–72)	2000	94 (88–100)
90	72 (71–73)	2500	96 (89–102)