Superior mesenteric artery calcification is associated with cardiovascular risk factors, systemic calcified atherosclerosis, and increased mortality

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ABSTRACT

Background: Atherosclerosis is a major risk factor for morbidity and mortality. However, epidemiologic data are sparse regarding risk factors for superior mesenteric artery calcification (SMAC), the association between SMAC and disease in other arterial beds, or the independent contribution of SMAC to risk of mortality. The objective of this study was to test the hypothesis that presence and extent of SMAC are associated with cardiovascular disease (CVD) risk factors, calcification in other arterial beds, and both cardiovascular and all-cause mortality, independent of classic risk factors and calcification in other arterial beds.

Methods: Arterial calcification in the superior mesenteric artery, celiac trunk, coronaries, thoracic aorta, abdominal aorta, and iliac arteries was evaluated by computed tomography in adults with no known CVD. Multiple logistic regression models examined risk factor associations for SMAC and SMAC as a risk factor for calcification in other arterial beds. Cox models were used to examine the association between SMAC and mortality.

Results: The average age of subjects was 56 years; 43.7% (1877/4300) were women, and 6.7% (290) had SMAC. Age (odds ratio [OR], 1.09; 95% confidence interval, 1.06-1.11), male sex (OR, 1.79; 95% CI, 1.08-3.03), dyslipidemia (OR, 1.38; 95% CI, 1.01-1.88), and any smoking (OR, 1.60; 95% CI, 1.20-2.14) were associated with SMAC presence. Notably, body mass index, body fat percentage, hypertension, diabetes, and family history of coronary heart disease were not significant risk factors for the presence of SMAC. SMAC presence was associated with calcification in all five other arterial beds (OR, 6.02; 95% CI, 3.76-9.66). During a median follow-up time of 9.4 years, there were 234 deaths, 76 of which were CVD related. SMAC extent (represented as per-unit increase in log [SMAC score + 1]; OR, 1.31; 95% CI, 1.01-1.71) was significantly associated with CVD mortality after full adjustment for risk factors and calcification in other arterial beds. SMAC presence (OR, 1.52; 95% CI, 1.10-2.12) and extent (OR, 1.25; 95% CI, 1.06-1.48) were also both significantly associated with all-cause mortality after full adjustment.

Conclusions: SMAC is associated with specific CVD risk factors as well as with calcification in all other arterial beds. SMAC extent was significantly associated with incident cardiovascular mortality, whereas both SMAC presence and extent were significantly associated with all-cause mortality, even after adjustment for risk factors and calcification in other arterial beds. Further studies are needed to determine whether SMAC is simply a marker for advanced and systemic disease or whether it confers increased mortality risk through an independent mechanism. (J Vasc Surg 2017;■:1-7.)

Subclinical atherosclerosis can be manifested as calcification in several arterial beds, even in individuals who are not traditionally defined as high risk.[1] Whereas calcification in vessels such as the coronary and renal arteries has been shown to have unique risk factors and additional contribution to mortality,[2,3] few data are available for the superior mesenteric artery (SMA).

The SMA arises from the abdominal aorta just distal to the celiac trunk and supplies the small intestine, part of the large intestine, and the pancreas. Presumably, SMA calcification (SMAC) represents progression of the same disease process that leads to atherosclerosis and calcified disease in other systemic arterial beds. Indeed, hyperlipidemia, diabetes, and smoking, all of which are well-established risk factors for atherosclerosis, are also risk factors for chronic mesenteric ischemia.[4] In this regard, specific radiographic qualitative and quantitative studies would further strengthen these associations or perhaps reveal additional prognostic value of the finding of SMAC for incident cardiovascular events and mortality.

This study tested the hypothesis that SMAC presence and extent are positively associated with cardiovascular disease (CVD) risk factors, calcified atherosclerosis in other arterial beds, and both cardiovascular and all-cause mortality, even after adjusting for risk factors and presence of systemic atherosclerotic disease.

METHODS

Subjects. From March 1, 2000, to July 3, 2003, 5156 healthy individuals without known CVD presented for preventive medicine services at a university-affiliated

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disease prevention center in San Diego, California. Of these, 4300 had complete survey and vascular calcification data and were included in our analysis. Subjects were self-referred or referred by a physician to obtain preventive health information and testing. Subjects completed a health history questionnaire that included information on history of hypertension, diabetes, high cholesterol, smoking (ever or never smoker), medications, and family history of coronary heart disease (CHD). The protocol for this study was approved by the Human Research Protection Program at the University of California, San Diego, and all subjects gave informed consent.

Laboratory measurements. Total, high-density, and low-density cholesterol and glucose measurements were obtained by fingerstick using the Cholestech LDX system (Cholestech, Hayward, Calif). Individuals with a total/high-density lipoprotein cholesterol ratio >5 or who reported using a medication to treat high cholesterol levels were classified as dyslipidemic. Diabetes was defined by use of prescribed diabetes medications at the time or a random glucose level >200 mg/dL. While seated, blood pressure was measured in the right arm by automated oscillometry after 5 minutes at rest. Hypertension was defined as a systolic pressure or diastolic pressure >140 or 90 mm Hg, respectively, or a self-reported history of physician-diagnosed hypertension *and* current use of an antihypertensive medication.

Imaging. Noncontrast-enhanced computed tomography imaging was conducted using an Imatron C-150 scanner (General Electric, San Francisco, Calif). Images for each arterial bed were obtained from the base of the skull to the pubic symphysis. Each bed was obtained by a distinct scan of the segment in question by using slice thicknesses of 3 mm for the coronary bed, 5 mm for the thorax, and 6 mm through the neck, abdomen, and pelvis. Imaging of the heart, thorax, and abdomen was conducted during separate breath holds at 50% maximal inspiration. These images were subsequently analyzed for the presence and extent of vascular calcification in the coronary arteries, thoracic aorta, abdominal aorta, SMA, celiac trunk, and iliac arteries. Quantitative calcium scores for each arterial bed were determined according to the method described by Agatston et al.[5] Data on a subgroup of these individuals have been previously reported.[3]

The SMA was identified at its anterior takeoff from the aorta and was tracked caudally until no longer discernible. In cases in which calcification extended from the aorta into the ostia of the SMA, a vertical plane simulating the wall of the aorta traversing the ostium was drawn to approximate the lumen of the aorta. Any calcification medial to the plane was excluded from the calculation as SMAC.

ARTICLE HIGHLIGHTS

- **Type of Research:** Retrospective, single-center, cross-sectional cohort study
- **Take Home Message:** In 4300 patients without cardiovascular disease, 6.7% had computed tomography evidence of superior mesenteric artery calcification, which was associated with cardiovascular-related and all-cause mortality.
- **Recommendation:** This study suggests that calcification of the superior mesenteric artery is associated with cardiovascular risk factors and is an independent risk factor for cardiovascular-related and all-cause mortality.

Mortality data. All-cause mortality was determined up to December 31, 2010, using Social Security Death Index searches and the National Death Index. Date and cause of death were extracted from death certificates and by the National Death Index search. The cause of mortality was ascertained by physician adjudicators (M.A.A., M.H.C.).

Statistical analysis. Baseline characteristics stratified by SMAC absence vs presence were examined using χ^2 analysis or Welch unequal variances *t*-test as appropriate. Proportions of calcification in other arterial beds were compared between those with and without SMAC using χ^2 analysis. Age- and sex-adjusted Spearman rank correlation was used to determine partial coefficient correlations of the extent of calcification (represented as $\log_{10}$ [score + 1]) between the SMA and the non-SMA arterial beds included in the analysis (the thoracic aorta, abdominal aorta, coronary arteries, iliac arteries, and celiac trunk).

As the prevalence of SMAC did not exceed 10% in our study sample, a multiple logistic regression model was used to determine risk factor associations for SMAC presence. The risk factors included were age, sex, body mass index (BMI), body fat percentage (BF%), dyslipidemia, any smoking, hypertension, diabetes, and family history of CHD. The model was then further adjusted for calcification in five other arterial beds (abdominal aorta, thoracic aorta, coronary arteries, iliac arteries, and celiac trunk). A similar analysis was performed using multiple linear regression, with SMAC extent (represented as $\log_{10}$ [SMAC score + 1]) as the outcome variable. In a separate logistic regression model, SMAC presence was used as a predictor for systemic calcified atherosclerosis, defined as having calcification in all five of the aforementioned non-SMA arterial beds. The model was first adjusted for age and sex and then for all risk factors. A similar analysis using SMAC extent rather than presence as a predictor was performed.

Survival analysis was performed using Kaplan-Meier curves and Cox regression models. SMAC presence and

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