Patterns and Risk Factors for Systemic Calcified Atherosclerosis

Matthew A. Allison, Michael H. Criqui, C. Michael Wright

Objective—Complex atherosclerotic lesions contain radio-opaque calcium hydroxyapatite deposits with the degree of calcification correlating with the extent of atherosclerosis. In this study, we aim to determine the patterns of systemic atherosclerotic calcification.

Methods and Results—Whole-body electron beam computed tomography scans were performed on 650 asymptomatic subjects to assess the carotid, coronary, proximal, and distal aorta and iliac vessels for atherosclerotic calcification. The mean age was 57.3 and 53% were male. Correlation patterns were similar in both genders, with the largest interbed correlations between the distal aorta and iliac vessels (r=0.51 to 0.60). The average man and woman had calcium earliest in the coronaries (younger than age 50 years) and the distal aorta (age 50 to 60), respectively. The prevalence of calcium was greater than 80% for most beds in men older than age 70 and greater than 60% in all beds for women. Approximately on third of subjects younger than 50 were free of calcified disease, whereas all subjects older than 70 were found to have some calcium. Age and hypertension were the dominant risk factors for systemic calcified atherosclerosis.

Conclusions—This study confirms that there are significant correlations and risk factor associations for calcified atherosclerosis in different vascular beds. (Arterioscler Thromb Vasc Biol. 2004;24:331-336.)

Key Words: calcium ■ atherosclerosis ■ electron beam computed tomography ■ coronary

Atherosclerosis begins in childhood and progresses from fatty streaks to raised lesions in adolescence and young adulthood.1–3 Raised lesions further progress to mature atheroma and complex lesions later in adulthood. Complex lesions and many atheroma contain radio-opaque calcium hydroxyapatite deposits,4,5 and the degree of calcification in arteries appears to correlate highly with the extent of atherosclerosis.6 Calcified plaques have been detected throughout the vasculature using computed tomography.7 Electron beam computed tomography is a noninvasive, reproducible8 screening procedure used to detect coronary calcification. Coronary calcification has been shown to be directly related to the severity and extent of underlying coronary plaque burden.9 Histopathologic research has also shown a high correlation between the extent of coronary calcification and total coronary atherosclerotic plaque burden.10 These correlations (r=0.90) have been shown to be true for all ages and for both sexes.11

The purpose of this study was to determine the correlations and patterns of calcified atherosclerosis in 5 different vascular beds as well as the relationship of atherosclerosis in these beds with traditional cardiovascular risk factors.

Methods

Subjects
From February 2001 to May 2002, 650 consecutive asymptomatic subjects who presented for preventive medicine services at a university-affiliated disease prevention center in San Diego, California, were evaluated for the extent of calcified atherosclerosis in 5 different vascular beds: carotid, coronary, proximal aorta, distal aorta, and iliac vessels. Most subjects were self-referred or referred by their primary care provider.

All subjects completed a detailed health history questionnaire before undergoing the scanning procedure. Smoking status was defined as current, former, or never, and it was then dichotomized to ever versus never. Diagnoses of hypertension, hyperlipidemia, and diabetes were identified by self-report and current use of anti-hypertensive, cholesterol-lowering, or anti-glycemic medications, respectively.

Imaging

All patients underwent imaging with an Imatron C-150 scanner. All images were obtained during a single session using 100-millisecond scan time and preceding caudally from the base of the skull to the symphysis pubis. Each bed was obtained by a distinct scan of the segment in question using the following slice thicknesses: 3 mm for the coronary bed; 6 mm through the neck, abdomen, and pelvis; and 5 mm for the thorax. Cardiac tomographic imaging was electrocardiographically triggered at 40% or 65% of the R-R interval, depending on the subject’s heart rate. Imaging of the heart, thorax, and abdomen was conducted during separate breath-holds at half-maximal inspiration.

The carotid and iliac beds comprised the right and left vessels of each of those segments. The coronary vascular bed comprised the left main, left anterior descending, left circumflex, and right coronary arteries. The proximal aortic bed was defined as the segment from the aortic root to the diaphragm, whereas the distal aortic bed was the segment from the diaphragm to the iliac bifurcation.
Atherosclerotic calcification was defined as a plaque of ≥2 pixels (area=0.67 mm²) with a density of ≥130 Hounsfield units. Quantitative calcium scores were determined according to the method described by Agatston et al. Calcium scores for vascular beds other than the coronaries were adjusted for slice thickness using the following formula: adjusted score=original score×slice thickness×3.0. Volume averaging was avoided by scoring each homogeneous slice-thickness segment separately. Calcium scoring of all beds was performed by one of us (M.A.A.) who had specific training for the methodology described.

Laboratory
All patients underwent serum lipid analysis obtained by fingerstick using the Cholestec LDX system. Body mass index was calculated with the patient lightly clothed (without shoes). Body fat measurement was conducted using bioimpedance on the Omron HBF-300.

Statistical Analysis
The subjects were analyzed as an entire group and by gender. Simple and age-adjusted correlations were calculated using the Spearman rank correlation procedure. The groups were then stratified by age into the following categories (in years): younger than 50, 50 to 59, 60 to 69, and 70 or older. Prevalences and median calcium scores were computed for each group and age category. Significant differences between the calcium scores within each bed were tested using the Kruskal-Wallis test.

Based on the results of the correlation analysis (Table 2), we defined 3 discrete calcification patterns: the left carotid and right carotid, the coronary bed, and the combination of the proximal aorta or distal aorta, or of the left iliac or right iliac. Thus, there were 8 possible patterns of vascular calcification: none; carotid alone (right, left, or both); coronary alone (left main, left anterior descending, left circumflex, right coronary artery); proximal/distal aorta or right/left iliacs (aorta/iliac); both carotid and coronaries (carotid–coronary); carotid and aorta or iliacs (carotid–aorta iliac); coronaries and aorta or iliacs (coronary–aorta iliac); and all carotid, coronary, and aorta iliacs) beds. These patterns were designed to represent regional anatomic areas and a gradation from localized to systemic disease. The prevalence within each pattern and age category was determined.

The potential association between the traditional cardiovascular risk factors and the presence and patterns of atherosclerotic calcification was explored by multivariable logistic regression. Risk factors that were associated with prevalent calcification at a significance level of 0.15 or less on univariate analysis were included in the final multivariable model. All statistical analyses were conducted using SAS version 8.0. The study protocol was approved by the Human Research Protection Program at the University of California at San Diego.

Results
The characteristics of the study subjects are presented in Table 1. Fifty-three percent of the sample was male and the average age was 57.6 years. Men were significantly different from women with respect to lipids, all body morphology measures, and the prevalence of hypercholesterolemia.

Two-hundred fifty-five subjects (39%) were found to be free of calcified atherosclerosis. There were significant differences between men and women with respect to the extent of calcification in all beds. Overall, in women the only bed with a non-zero median calcium score was the distal aorta (median calcium score=22), whereas in men only the carotid and proximal aortic areas had median scores of zero. The coronary, distal aortic, and iliac beds in men had median calcium scores of 24, 144, and 250, respectively.

Adjusted Spearman correlations are shown for women and men in Table 2. For the entire group, the range of age-adjusted correlations extended from 0.28 to 0.76, with the smallest correlation being between the coronary bed and the right carotid artery, whereas the largest was between the right and left iliac vessels. All the intrabed (ie, left carotid versus right carotid) correlations were moderate (r=0.56 to 0.76) except for the correlation between the proximal and distal aorta (0.45). The smallest interbed correlation was between the coronary and carotid beds (0.28 to 0.29), whereas the largest was found between the distal aorta and both iliac beds (0.58 to 0.59). The distal aorta had similar relationships with the other vascular beds, as did the right and left iliac arteries.

Table 3 shows the prevalence of atherosclerotic calcification per age category (younger than 50, 50 to 60, 60 to 70, older than 70) and vascular bed. In both groups, the coronary bed had the highest prevalence of disease at the earliest age (younger than 50). This pattern held in men for all age groups except for those in the 7th decade, when calcium was more prevalent in the distal aorta. In women after the age of 50, the distal aorta demonstrated the highest prevalence of disease until the 8th decade, when proximal aortic disease became most prevalent. The prevalence of calcium was >80% for most beds in men older than age 70 and >60% in all beds for women of the same age group.

The median calcium scores indicating the extent of calcification in the beds are also displayed in Table 3. The first median calcium score that was greater than zero was found in the distal aorta in women during the 6th decade and in the coronary bed in men younger than 50. Men also had significantly more calcified disease than women in all beds. In both genders, the extent of calcification increased exponentially with increasing age.

Figure 1A and B shows the prevalence of the atherosclerotic calcification patterns by age group for women and men, respectively. Women had a much higher prevalence of “zero”

TABLE 1. Descriptive Statistics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men (N=357)</th>
<th>Women (N=293)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age* (y)</td>
<td>57.3 (10.8)</td>
<td>58.0 (10.8)</td>
<td>0.47</td>
</tr>
<tr>
<td>HDL* (mg/dL)</td>
<td>44.7 (13.5)</td>
<td>63.1 (16.8)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>TC/HDL ratio*</td>
<td>4.6 (1.5)</td>
<td>3.5 (1.4)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Triglycerides* (mg/dL)</td>
<td>172.3 (98.8)</td>
<td>155.9 (98.5)</td>
<td>0.05</td>
</tr>
<tr>
<td>BMI* (kg/m²)</td>
<td>27.8 (3.9)</td>
<td>25.7 (4.8)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Total body fat* (%)</td>
<td>26.5 (5.5)</td>
<td>34.2 (7.2)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Visceral fat* (cm³)</td>
<td>104.8 (42.1)</td>
<td>54.5 (34.9)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>VF/BF ratio* (cm³/%)</td>
<td>3.9 (1.3)</td>
<td>1.7 (2.4)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Hypertension†</td>
<td>28</td>
<td>24.6</td>
<td>0.33</td>
</tr>
<tr>
<td>Hypercholesterolemia†</td>
<td>34.2</td>
<td>13.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Diabetes mellitus†</td>
<td>3.4</td>
<td>1.4</td>
<td>0.10</td>
</tr>
<tr>
<td>Current smoker†</td>
<td>7</td>
<td>7.9</td>
<td>0.68</td>
</tr>
<tr>
<td>Former smoker†</td>
<td>41.7</td>
<td>42</td>
<td>0.95</td>
</tr>
</tbody>
</table>

*Mean (SD). †Percent of sample.
HDL indicates high-density lipoprotein; TC, total cholesterol; BMI, body mass index; VF, visceral fat; BF, total body fat.
calcium before age 50 compared with men (53% versus 30%, respectively). In both genders, the prevalence of having no calcium decreased significantly until the 8th decade, when no subject was found to be free of calcified disease. However, the prevalence of calcium distributed diffusely ("all") increased in a nearly linear fashion in men and exponentially in women, whereby 82% of men and 67% of women older than age 70 exhibited this pattern. Before age 50, the pattern with the highest prevalence of calcium was the coronary bed (coronary) in men (26%), whereas the pattern aorta–iliac was similar to coronary in women at this age (20% versus 18.6%, respectively). Interestingly, no subject was found to have disease confined only to the coronary and carotid beds ("carotid–coronary").

The odds for the presence or absence of atherosclerotic calcification in each given vascular bed derived from multivariable logistic regression are presented in Table 4. Dashed lines indicate non-significant associations at a probability value of 0.15 on univariable analysis. Age per 10 years was a consistent predictor of disease in all beds, with largest odds ratios (OR) for the proximal aorta in both genders (6.1 in men and 7.0 in women). In men, hypertension was the next most prevalent predictor of disease, with significant OR in the carotid (3.2), coronary (3.9), and proximal aortic beds (2.7). In both genders, smoking status was significant for disease in the distal aorta and iliac vessels (OR range: 2.3 to 3.6). Although diabetes was related to calcification in univariable analysis, the increased OR for diabetes were not significant in the multivariable models, partly because of the low prevalence of this disease.

Table I (available online at http://atvb.ahajournals.org) shows the results for the multivariable analysis of patterns of atherosclerotic calcification in the carotid, coronary, and iliac beds in men and women.

| Table 2. Age-Adjusted Vascular Bed Correlations* |
|-----------------|----------------|-----------------|-----------------|----------------|----------------|----------------|
|                 | RCAR        | LCAR           | COR             | PAO            | DAO            | RIL            | LIL            |
| Women           |             |                |                 |                |                |                |                |
| RCAR            | 1.0         | 0.48           | 0.21            | 0.37           | 0.26           | 0.29           | 0.30           |
| LCAR            | 0.48        | 1.0            | 0.20            | 0.38           | 0.26           | 0.32           | 0.34           |
| COR             | 0.21        | 0.20           | 1.0             | 0.35           | 0.31           | 0.31           | 0.35           |
| PAO             | 0.37        | 0.38           | 0.35            | 1.0            | 0.46           | 0.43           | 0.42           |
| DAO             | 0.26        | 0.27           | 0.31            | 0.46           | 1.0            | 0.53           | 0.51           |
| RIL             | 0.29        | 0.32           | 0.31            | 0.43           | 0.53           | 1.0            | 0.73           |
| LIL             | 0.30        | 0.34           | 0.35            | 0.42           | 0.51           | 0.73           | 1.0            |
| Men             |             |                |                 |                |                |                |                |
| RCAR            | 1.0         | 0.61           | 0.31            | 0.42           | 0.40           | 0.30           | 0.38           |
| LCAR            | 0.61        | 1.0            | 0.32            | 0.38           | 0.42           | 0.35           | 0.42           |
| COR             | 0.31        | 0.32           | 1.0             | 0.35           | 0.41           | 0.46           | 0.44           |
| PAO             | 0.42        | 0.38           | 0.35            | 1.0            | 0.46           | 0.36           | 0.36           |
| DAO             | 0.40        | 0.42           | 0.41            | 0.46           | 1.0            | 0.60           | 0.59           |
| RIL             | 0.30        | 0.35           | 0.46            | 0.36           | 0.60           | 1.0            | 0.74           |
| LIL             | 0.38        | 0.42           | 0.44            | 0.36           | 0.59           | 0.74           | 1.0            |

*P<0.01 for all correlations.
RCAR indicates right carotid artery; LCAR, left carotid artery; COR, coronary; PAO, proximal aorta; DAO, distal aorta; RIL, right iliac; LIL, left iliac.

| Table 3. Prevalences and Median Calcium Scores Per Age Category and Vascular Bed** |
|-----------------|----------------|-----------------|-----------------|----------------|----------------|----------------|
| Age             | N‡            | RCAR           | LCAR           | COR             | PAO            | DAO            | RIL            | LIL            |
| Women           |               |                |                |                 |                |                |                |                |
| <50             | 34            | 0 [0]          | 0 [0]          | 27 [0]          | 14 [0]         | 16 [0]         | 14 [0]         | 10 [0]         |
| Men             |               |                |                |                 |                |                |                |                |
| <5              | 50            | 0 [0]          | 4 [0]          | 51 [1]          | 8 [0]          | 20 [0]         | 28 [0]         | 22 [0]         |

*Prevalence (%) [median calcium score].
†P<0.05 for all vascular beds.
‡Stratum size.
calcification. In men and women, 10-year increases in age were significantly associated with the presence of calcium for many of the vascular patterns when compared with the absence of calcification throughout the vasculature. In both sexes, the largest OR for this risk factor were found for diffuse (all group) calcification (OR=74.4 and 15.2 in women and men, respectively). The next strongest risk factor was a history of hypertension, which was significantly associated with aorta/iliac calcification in women (4.7) and coronary and aorta/iliac calcification (17.5), as well as diffuse calcification (21.2) in men. Cigarette smoking was significant associated with aorta/iliac calcification in women (4.7) and was a history of hypertension, which was significantly associated with the presence of calcium for many of the vascular patterns when compared with the absence of calcification throughout the vasculature. In both

Discussion

Correlations across vascular beds were similar in pattern for men and women, with men exhibiting larger values across most vascular beds. This finding likely represents the age of the cohort studied and the later appearance of calcified disease in women. A recent nomogram for coronary calcification demonstrated a 10- to 15-year lag for women compared with men with respect to the first appearance of atheromatous disease.12 In this report, the average age for the appearance of calcium was age younger than 40 in men and between 55 and 60 for women, which is a similar time interval as that found in the present study (Table 3).

Intrabed correlations were larger than interbed correlations, indicating that the extent of disease within a vascular bed is closely related to the anatomic origin of the vessel involved.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Carotids</th>
<th>Coronary</th>
<th>Proximal Aorta</th>
<th>Distal Aorta</th>
<th>Iliacs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (per 10 y)</td>
<td>5.9</td>
<td>2.2</td>
<td>6.1</td>
<td>5.5</td>
<td>2.9</td>
</tr>
<tr>
<td>Total body fat (5%)</td>
<td>0.9</td>
<td>1.1</td>
<td>0.9</td>
<td>0.6</td>
<td>2.7</td>
</tr>
<tr>
<td>BMI (3 units)</td>
<td>1.5</td>
<td>1.9</td>
<td>1.2</td>
<td>3.2</td>
<td>2.7</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>0.7</td>
<td>0.9</td>
<td>1.9</td>
<td>2.6</td>
<td>1.0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.5</td>
<td>1.0</td>
<td>1.9</td>
<td>2.6</td>
<td>0.5</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.5</td>
<td>1.0</td>
<td>1.9</td>
<td>2.6</td>
<td>1.0</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>1.5</td>
<td>1.0</td>
<td>1.9</td>
<td>2.6</td>
<td>1.0</td>
</tr>
<tr>
<td>FHx of PreCHD†</td>
<td>1.5</td>
<td>1.0</td>
<td>1.9</td>
<td>2.6</td>
<td>1.0</td>
</tr>
</tbody>
</table>

*Odds ratio.
†95% Confidence interval.
‡Family history of premature coronary heart disease.
One exception to this finding is the interbed correlations found between the distal aorta and iliac vessels. These correlations were of similar magnitude to those found for intrabed correlations, suggesting that these beds are developing atherosclerosis in a similar time frame and to a similar degree. During the scoring procedure, we noted that most of the plaque identified in the iliac circulation occurred just distal to the aortic bifurcation and was found in association with plaque just proximal to this point. Vessel bifurcations are areas that have been shown to be predisposed to development of atherosclerosis. Abrupt curvature of the vessel wall causes a disruption of laminar flow, resulting in a reduction in shear stress that stimulates the release of vasoactive substances, thereby raising the potential endothelial dysfunction.

In 1957, DeBakey et al first published the concept of the segmental nature of atherosclerotic vascular occlusive disease, and they have subsequently published several articles on the subject of systemic patterns of atherosclerosis. Similar to our findings, these studies revealed that men have a higher overall prevalence of atherosclerotic disease compared with women. However, they did not find a differential predilection for initial manifestation of disease among the defined patterns, whereas we found a definite propensity for earlier expression of disease in the coronary bed followed by the distal aorta. Probable explanations for this disparity are the difference in study populations (surgical versus unscreened patients) and methodology for determining the location and extent of disease (angiography versus vascular calcium).

Our study demonstrates that the extent of calcium caused by atherosclerosis increases with age throughout the vasculature. Less than 5% of men and women younger than 50 had diffuse disease (all), whereby after age 70 more than two thirds of men and women had disease in all of the 5 vascular beds that were studied. These results are supported by a study by Khoury et al, who studied 102 subjects with significant angiographic coronary artery disease (CAD) for the presence of systemic atherosclerosis using ultrasonography (intimal medial thickness >1 mm). They found that patients with CAD had a significantly higher risk of having atherosclerotic plaques in the peripheral vessels compared with those without CAD. Extracoronary plaque was also found to be a stronger predictor of CAD than conventional risk factors.

Kuller et al previously reported that aortic calcium occurs 6- to 10-times more often than that of the coronaries at any given age. Similarly, our study found a higher prevalence of and median score for calcium in the aorta compared with the coronaries for all ages in both genders, except for men younger than 50. The findings from the multivariable risk factor analyses (Tables 4 and 1) expand on these results, whereby there was a larger magnitude of association between atherosclerotic burden in the more peripheral circulation (ie, aorta and iliacs) and aging than that found for the coronary bed. Furthermore, increasing age was strongly associated with more diffuse patterns of disease after adjusting for risk factors.

Our results also suggest that atherosclerosis may develop in woman at an accelerated rate after menopause. In our study, 47% of women who were younger than 50, compared with 70% of men of this age, were found to have calcium. This prevalence increased to 73% between ages 50 to 60 years and to 91% between ages 60 and 70 years. The corresponding prevalences in men were 92% and 98%, respectively, equating to a 44% increase in women compared with a 28% increase in men over the same time frame. These results suggest a protective effect of the hormones associated with menopause for the development of atherosclerosis. However, the Heart and Estrogen/Progesterin Replacement Study and the Estrogen Replacement and Atherosclerosis Trial did not find significant differences in rates of progression for atherosclerosis by carotid intimal medial thickness and coronary angiography, respectively, in women who were provided with hormone therapy versus those who used placebo. Importantly, the mean age of women in these studies was significantly older than that of women in our study (67 and 65.8, respectively), whereas the follow-up was <4 years for both. Longer prospective studies are needed to clarify these differences.

Previous studies have shown that individuals with claudication or subclinical peripheral arterial disease are at increased risk for cardiovascular mortality and events. CAD patients with stable disease or who are status-post coronary artery bypass surgery and who also have concomitant proximal artery disease have mortality rates that are 25% and 71% higher, respectively, than those who do not. Given these results, some authors have suggested that a noninvasive assessment of the peripheral circulation should be included in the evaluation of patients with chest pain. Furthermore, the 34th Bethesda Conference reported that non-invasive assessment for subclinical disease is appropriate for individuals with intermediate risk for coronary heart disease. Data on the relationship between cardiovascular disease events and atherosclerotic calcification are limited to the coronary circulation. Studies assessing the potential association between subclinical disease in different vascular areas and events are needed to confirm that early detection can effectively risk-stratify individual patients.

Acknowledgment
This work was supported in part by the American Heart Association project number 0325002Y.

References


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Arterioscler Thromb Vasc Biol. 2004;24:331-336; originally published online December 4, 2003;
doi: 10.1161/01.ATV.0000110786.02097.0c
Arteriosclerosis, Thrombosis, and Vascular Biology is published by the American Heart Association, 7272
Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1079-5642. Online ISSN: 1524-4636

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
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### Table I – Odds for Atherosclerotic Calcification Pattern per Risk Factor

<table>
<thead>
<tr>
<th>Women Variable</th>
<th>Any</th>
<th>Carotids Alone</th>
<th>Coronaries Alone</th>
<th>Aorta/Iliacs Alone</th>
<th>Carotids &amp; Coronaries</th>
<th>Carotids &amp; Aorta/Iliacs</th>
<th>Coronaries &amp; Aorta/Iliacs</th>
<th>All</th>
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</thead>
<tbody>
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<td>Age (10 years)</td>
<td>5.0</td>
<td>2.5-10.0</td>
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<td>- -</td>
<td>3.7</td>
<td>2.0-6.7</td>
<td>47.7</td>
<td>5.4-423.6</td>
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<tr>
<td>Total Body Fat (5%)</td>
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<td>0.4-1.3</td>
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<td>1.0</td>
<td>0.7-1.3</td>
<td>- -</td>
<td>0.7</td>
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<tr>
<td>BMI (3 units)</td>
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<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>-</td>
<td>2.2</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>1.1</td>
<td>0.3-5.0</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>5.3</td>
<td>0.4-68.0</td>
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<tr>
<td>Hypertension</td>
<td>5.0</td>
<td>1.4-10.0</td>
<td>- -</td>
<td>2.9</td>
<td>0.7-12.3</td>
<td>4.7</td>
<td>1.4-15.8</td>
<td>3.7</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Cigarette Smoking</td>
<td>2.5</td>
<td>1.1-5.0</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>- -</td>
<td>-</td>
<td>6.0</td>
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<tr>
<td>FHx of PreCHD</td>
<td>2.0</td>
<td>0.8-5.0</td>
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<td>2.3</td>
<td>0.7-6.9</td>
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<td>0.6-5.1</td>
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<th>Men Variable</th>
<th>Any</th>
<th>Carotids Alone</th>
<th>Coronaries Alone</th>
<th>Aorta/Iliacs</th>
<th>Carotids &amp; Coronaries</th>
<th>Carotids &amp; Aorta/Iliacs</th>
<th>Coronaries &amp; Aorta/Iliacs</th>
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<tr>
<td>Age (10 years)</td>
<td>5.0</td>
<td>2.5-10.0</td>
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<td>- -</td>
<td>4.0</td>
<td>1.8-9.0</td>
<td>8.8</td>
<td>2.1-37.8</td>
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<tr>
<td>Total Body Fat (5%)</td>
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<td>0.9-1.7</td>
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<td>1.3</td>
<td>0.8-2.2</td>
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<td>0.8-4.8</td>
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<tr>
<td>BMI (3 units)</td>
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<td>- -</td>
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<tr>
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<td>0.1-8.0</td>
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<tr>
<td>FHx of PreCHD</td>
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</tbody>
</table>

*Odds Ratio, †95% Confidence Interval, ‡Family History of Premature CHD