Prevalence of Coronary Atherosclerosis in Patients With Cerebral Infarction

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Background and Purpose—There is an overlap between stroke and coronary heart disease, but the exact prevalence of coronary artery disease in patients with nonfatal cerebral infarction is unclear, particularly when there is no known history of coronary heart disease.

Methods—We consecutively enrolled 405 patients presenting with acute cerebral infarction documented by neuroimaging who underwent carotid and femoral artery, thoracic, and abdominal aorta ultrasound examinations. Of the 342 patients with no known coronary heart disease, 315 underwent coronary angiography a median of 8 days (interquartile range, 6–11) after stroke onset.

Results—Coronary plaques on angiography, regardless of stenosis severity, were present in 61.9% of patients (95% confidence interval [CI], 56.5–67.3) and coronary stenoses ≥50% were found in 25.7% (95% CI, 20.9–30.5). The overall prevalence of coronary plaque increased with the number of arterial territories (carotid or femoral arteries) involved, with an adjusted odds ratio of coronary artery disease of 1.25 (95% CI, 1.92–9.68) for presence of plaque in 1 territory, and 4.31 (95% CI, 1.92–9.68) for presence of plaque in both territories, compared with no plaque in either territory. The presence of plaque in both femoral and carotid arteries had an age- and sex-adjusted positive predictive value of 84% for presence of coronary plaque and a negative predictive value of 44%.

Conclusions—There is a high burden of silent coronary artery disease in patients with nonfatal cerebral infarction and no known coronary heart disease, even in the absence of systemic atherosclerosis. The prevalence is even higher in patients with evidence of carotid and/or femoral plaque. (Stroke. 2011;42:22-29.)

Key Words: atherosclerosis • cerebral infarction • coronary arteries • myocardial infarction

In the early years after a stroke, the most common vascular event is another stroke.1 However, at 5 years there are twice as many deaths from myocardial infarction as there are from recurrent stroke.2,3 Among 846 ischemic stroke patients included in the Virtual International Stroke Trials Archive, 35 (4.1%) died from cardiac causes at 12 weeks.4 Although this risk is well-documented in patients with stroke5–7 and in subjects with carotid atherosclerosis,8 how much of it can be predicted from medical history or from the presence of arterial plaque in extracardiac locations (such as carotid or femoral arteries) and how much is related to “silent” asymptomatic coronary artery disease is unknown. The exact prevalence of silent coronary atherosclerosis in stroke patients with no known coronary heart disease is not known. One autopsy series found a high prevalence of coronary artery disease in patients with fatal cerebral infarction, which was even higher in the presence of carotid or cerebral artery atherosclerosis.9 However, there has been no systematic angiography evaluation of the prevalence of coronary artery disease in a large group of consecutive patients with nonfatal cerebral infarction with no known history of coronary heart disease. Our aim was to describe the prevalence of coronary artery disease in a large cohort of consecutive patients with nonfatal cerebral infarction, and...
to assess whether the presence of systemic atherosclerosis in extracardiac locations affected this prevalence.

**Patients and Methods**

**Study Population**

The Asymptomatic Myocardial Ischemia in Stroke and Atherosclerotic Disease (AMISTAD) study was a prospective, single-center registry. All consecutively enrolled patients 18 years of age or older admitted with an acute ischemic stroke documented by neuroimaging and a Rankin scale <5 were offered the opportunity to participate and were enrolled within 10 days of symptom onset after providing written informed consent. Pregnant women, patients with other nonvascular diseases associated with a life expectancy of <30 months, and patients with cerebral infarction attributable to carotid or vertebral artery dissection or secondary to a revascularization procedure were excluded. The research protocol was approved by the Ethics Committee of Paris Bichat-Claude Bernard (2004/11, May 9, 2004) and of Ile de France Number 1 Hotel Dieu (amendment 1, 0611369, September 14, 2006; and amendment 2, 0611445, 24/11/2006).

**Evaluation of Atherosclerotic Disease Burden**

All patients had a standardized evaluation. Carotid atherosclerosis was evaluated by ultrasound examination of both extracranial carotid arteries. Patients were categorized as having either a normal carotid artery wall or an atherosclerotic plaque with or without stenosis. We used the term “plaque” to describe the anatomic lesion of an artery produced by atherosclerotic disease, regardless of whether they induced an arterial stenosis or a lumen narrowing. Stenoses were measured on horizontal cross-sections based on residual lumen relative to the external diameter of the artery. Coronary atherosclerosis was evaluated by coronary angiography in patients with no known history of coronary heart disease (history of acute coronary syndrome, myocardial infarction, or previous coronary revascularization). Results were categorized into absence of plaque, plaque with or without stenosis of any degree, stenosis 1% to 49%, 50% to 69%, 70% to 99%, and 100% (total occlusion). Patients who had a known history of coronary heart disease did not undergo coronary angiography but were grouped in the analyses with patients with coronary plaque. Atherosclerosis of the aorta was evaluated by transesophageal echocardiography (aortic arch and thoracic descending aorta) and abdominal ultrasound (abdominal aorta, including measurement of infrarenal aortic diameter). Femoral atherosclerosis was evaluated by ultrasonography. Results of the aortic arch examination were categorized into absence of plaque, plaque <4 mm in thickness, or plaque ≥4 mm.

**Risk Factor Evaluation**

All patients had an evaluation of their demographic characteristics and risk factors after a face-to-face interview and using a structured questionnaire. Blood samples were collected in fasting conditions at risk factors after a face-to-face interview and using a structured questionnaire. Blood samples were collected in fasting conditions at and were enrolled within 10 days of symptom onset after providing written informed consent. Pregnant women, patients with other nonvascular diseases associated with a life expectancy of <30 months, and patients with cerebral infarction attributable to carotid or vertebral artery dissection or secondary to a revascularization procedure were excluded. The research protocol was approved by the Ethics Committee of Paris Bichat-Claude Bernard (2004/11, May 9, 2004) and of Ile de France Number 1 Hotel Dieu (amendment 1, 0611369, September 14, 2006; and amendment 2, 0611445, 24/11/2006).

**Table 1. Prevalence of Asymptomatic Coronary Artery Disease by Site and Severity Among 315 Patients Who Underwent Coronary Angiography**

<table>
<thead>
<tr>
<th>Site</th>
<th>Degree of Stenosis</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>None</td>
</tr>
<tr>
<td>Any artery, N (%)</td>
<td>120 (38.1)</td>
</tr>
<tr>
<td>Left main coronary artery, N (%)</td>
<td>271 (86.0)</td>
</tr>
<tr>
<td>Anterior interventricular artery, N (%)</td>
<td>158 (50.2)</td>
</tr>
<tr>
<td>Circumflex artery, N (%)</td>
<td>193 (61.3)</td>
</tr>
<tr>
<td>Right coronary artery, N (%)</td>
<td>169 (53.6)</td>
</tr>
</tbody>
</table>

**Statistical Analysis**

All statistical tests performed with the use of SAS software, version 9.1, were performed at the 2-tailed level of 0.05. Data are presented as mean (standard deviation) for continuous variables and percentage (count) for dichotomous variables. Among the overall study group, we studied the independent association of conventional coronary risk factors with coronary artery disease (plaques with or without stenosis, and stenoses ≥50%) using multiple logistic regression analysis. A modified Framingham global risk score was determined using history of treated hypertension for the subset of 289 patients aged 30 to 74 years who had complete information on score components (age, gender, hypertension, total cholesterol, high-density lipoprotein cholesterol, current smoking, diabetes). We calculated the odds ratio (OR) of coronary artery disease per each point score increase in the Framingham global risk score using logistic regression analysis. We investigated the association of coronary artery disease with presence of plaques in the carotid artery, femoral artery, and aorta (ascending thoracic, and abdominal aorta) using logistic regression analysis adjusted for age and gender. An additional adjustment was performed by including risk factors associated with the presence of coronary artery disease (hypertension, dyslipidemia, current smoking, and family history of coronary heart disease) into the model. Because of the significant association of coronary artery disease with carotid and femoral plaques, we studied the joint effect of carotid and femoral plaques and computed the OR of coronary artery disease for presence of plaque in 1 arterial territory and in both arterial territories using patients with no plaque in carotid and femoral arteries as the control group. Finally, we...
Between June 2005 and December 2008, 785 patients were enrolled, of whom 15.6% (N = 120) had a known history of coronary heart disease. The demographic and clinical characteristics of the 378 patients who underwent coronary angiography or had a known history of coronary heart disease are described in Table 2. They were categorized according to the results of coronary angiography (no disease, silent stenosis <50%, and silent stenosis ≥50%) or to the presence of known history of coronary heart disease.

Table 2. Demographics and Clinical Characteristics of 378 Study Patients According to Coronary Artery Disease Subgroup

<table>
<thead>
<tr>
<th>Coronary Artery Disease Subgroups</th>
<th>Absence (N=120)</th>
<th>Silent Stenosis &lt;50% (N=114)</th>
<th>Silent Stenosis ≥50% (N=81)</th>
<th>Known Coronary Heart Disease (N=127)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>55.4 ± 13.2</td>
<td>62.8 ± 12.1</td>
<td>67.4 ± 10.3</td>
<td>67.6 ± 63</td>
</tr>
<tr>
<td>Men, N (%)</td>
<td>80 (66.7)</td>
<td>86 (75.4)</td>
<td>62 (76.5)</td>
<td>56 (88.9)</td>
</tr>
<tr>
<td>Body mass index, kg/m</td>
<td>25.9 ± 4.2</td>
<td>26.4 ± 4.9</td>
<td>26.0 ± 4.6</td>
<td>25.7 ± 3.8</td>
</tr>
<tr>
<td>Hypertension, N (%)</td>
<td>75 (62.5)</td>
<td>96 (84.2)</td>
<td>78 (96.3)</td>
<td>60 (95.2)</td>
</tr>
<tr>
<td>Diabetes, N (%)</td>
<td>26 (21.7)</td>
<td>25 (21.9)</td>
<td>18 (22.2)</td>
<td>16 (25.4)</td>
</tr>
<tr>
<td>Dyslipidemia, N (%)</td>
<td>36 (30.0)</td>
<td>39 (34.2)</td>
<td>40 (49.4)</td>
<td>48 (76.2)</td>
</tr>
<tr>
<td>Current smoker, N (%)</td>
<td>39 (32.5)</td>
<td>53 (46.5)</td>
<td>34 (42.0)</td>
<td>30 (32.3)</td>
</tr>
<tr>
<td>Personal history of stroke, N (%)</td>
<td>6 (5.0)</td>
<td>10 (8.8)</td>
<td>7 (8.6)</td>
<td>8 (12.9)</td>
</tr>
<tr>
<td>Family history of coronary heart disease, N (%)</td>
<td>24 (20.0)</td>
<td>27 (23.9)</td>
<td>23 (28.4)</td>
<td>22 (35.5)</td>
</tr>
<tr>
<td>Family history of stroke, N (%)</td>
<td>31 (25.8)</td>
<td>29 (25.4)</td>
<td>14 (17.3)</td>
<td>13 (21.0)</td>
</tr>
<tr>
<td>Framingham point scores*</td>
<td>6.4 ± 3.7</td>
<td>8.7 ± 2.9</td>
<td>9.6 ± 2.5</td>
<td>8.0 ± 3.0</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

*Available for 289 patients aged 30 to 74 years who had complete information on score components.
coronary heart disease) and with the presence of coronary stenosis ≥50% (ie, silent stenosis ≥50% or known coronary heart disease; Figure 2). Consistent results were found when multivariable analysis was repeated in the subset of 315 patients with no known coronary heart disease (Figure 2).

Among patients aged 30 to 74 years, increasing Framingham risk scores were associated with the presence of coronary plaque (OR per point increase, 1.27; 95% CI, 1.17–1.38) and coronary stenosis ≥50% (OR, 1.15; 95% CI, 1.06–1.25). Restricting the analysis to patients with no known coronary heart disease yielded consistent results (OR for coronary plaque, 1.30; 95% CI, 1.19–1.43; OR for coronary stenosis ≥50%, 1.24; 95% CI, 1.12–1.38).

**Figure 2.** Independent associations of vascular risk factors with (A) coronary plaque and with (B) coronary stenosis ≥50%. Coronary plaque defined as silent plaque regardless of stenosis degree or known coronary heart disease. Coronary stenosis ≥50% defined as silent plaque with arterial lumen reduction ≥50% or known coronary heart disease. Black squares indicate multivariable-adjusted odds ratios (OR) computed in all study patients (N=378), and gray squares indicate the multivariable-adjusted OR computed in patients with no known coronary heart disease (N=315).

**Coronary Artery Disease and Burden of Systemic Atherosclerosis**

Among 378 patients, 98.7% (N=373) underwent carotid ultrasonography examination; 93.4% of patients (N=353) had a femoral examination, 89.4% (N=338) had an abdominal aorta examination, and 79.1% (N=299) had transesophageal echocardiography examination of the thoracic aorta. Table 3 shows the distribution of coronary artery disease according to the presence or absence of atherosclerotic plaque in each of these arterial territories. After adjustment for age and gender, the prevalence of coronary plaque was higher in the presence than in the absence of atherosclerotic plaque in the carotid artery (78% vs 58%; P<0.001), femoral artery (78% vs 46%; P<0.001), abdominal aorta (79% vs 66%; P=0.06), aortic arch (75% vs 63%; P=0.04), and descending thoracic aorta (80% vs 66%; P=0.05).

In multivariable analysis, including age, gender, hypertension, dyslipidemia, current smoking, and family history of coronary heart disease, the presence of coronary plaque was significantly associated with the presence of plaque in the carotid and femoral arteries (Figure 3A). Similar results were
found when restricting the analysis to coronary stenoses \( \geq 50\% \) (Figure 3B) or to patients with no known coronary heart disease (Figures 3A, B). We therefore studied the interrelation between carotid, femoral, and coronary artery disease (Figure 4). The prevalence of coronary plaque and prevalence of coronary stenosis \( \geq 50\% \) were markedly increased when plaques were present in both territories. The age- and gender-adjusted positive and negative predictive values of the presence of atherosclerotic plaque in both territories for predicting the presence of coronary plaque were 84\% and 44\%, respectively. The corresponding adjusted predictive values for the presence of coronary stenoses \( \geq 50\% \) were 45\% and 74\%, respectively. After adjustment for risk factors for coronary artery disease, the OR for having coronary plaque was 1.25 (95\% CI, 0.58–2.71) for patients with plaque in 1 extracardiac arterial location and 4.31 (95\% CI, 1.92–9.68) for those with both extracardiac arterial locations compared to patients without evidence of extracardiac arterial disease. The corresponding adjusted OR of having a coronary stenosis were 1.57 (95\% CI, 0.57–4.34) and 2.68 (95\% CI, 1.00–7.16), respectively. Similar adjusted OR were found in an analysis restricted to patients with no known coronary heart disease (data not shown).

Table 3. Distribution of Coronary Artery Disease Subgroups According to Presence or Absence of Plaque in Other Arterial Territories

<table>
<thead>
<tr>
<th>Coronary Artery Disease Subgroups</th>
<th>N</th>
<th>Absence</th>
<th>Silent Stenosis &lt;50%</th>
<th>Silent Stenosis ( \geq 50% )</th>
<th>Known Coronary Heart Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extracranial carotid artery, N (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absence</td>
<td>115</td>
<td>61 (53.1)</td>
<td>32 (27.8)</td>
<td>12 (10.4)</td>
<td>10 (8.7)</td>
</tr>
<tr>
<td>Presence</td>
<td>258</td>
<td>54 (20.9)</td>
<td>82 (31.8)</td>
<td>69 (26.7)</td>
<td>53 (20.5)</td>
</tr>
<tr>
<td>Aortic arch, N (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absence</td>
<td>143</td>
<td>66 (46.2)</td>
<td>38 (26.6)</td>
<td>19 (13.3)</td>
<td>20 (14.0)</td>
</tr>
<tr>
<td>Presence</td>
<td>156</td>
<td>34 (21.8)</td>
<td>57 (36.5)</td>
<td>38 (24.4)</td>
<td>27 (17.3)</td>
</tr>
<tr>
<td>Descending aorta, N (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absence</td>
<td>230</td>
<td>89 (38.7)</td>
<td>74 (32.2)</td>
<td>39 (17.0)</td>
<td>28 (12.2)</td>
</tr>
<tr>
<td>Presence</td>
<td>69</td>
<td>11 (15.9)</td>
<td>21 (30.4)</td>
<td>18 (26.1)</td>
<td>19 (27.5)</td>
</tr>
<tr>
<td>Abdominal aorta, N (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absence</td>
<td>247</td>
<td>97 (39.3)</td>
<td>75 (30.3)</td>
<td>38 (15.4)</td>
<td>37 (15.0)</td>
</tr>
<tr>
<td>Presence</td>
<td>91</td>
<td>15 (16.5)</td>
<td>25 (27.5)</td>
<td>35 (38.4)</td>
<td>16 (17.6)</td>
</tr>
<tr>
<td>Femoral artery, N (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absence</td>
<td>93</td>
<td>60 (64.5)</td>
<td>19 (20.4)</td>
<td>9 (9.7)</td>
<td>5 (5.4)</td>
</tr>
<tr>
<td>Presence</td>
<td>260</td>
<td>54 (20.8)</td>
<td>88 (33.8)</td>
<td>68 (26.2)</td>
<td>50 (19.2)</td>
</tr>
</tbody>
</table>

Figure 3. Association between systemic atherosclerosis burden and coronary plaque (A) and coronary stenosis \( \geq 50\% \) (B). Coronary plaque defined as silent plaque regardless of stenosis degree or known coronary heart disease. Coronary stenosis \( \geq 50\% \) defined as silent plaque with arterial lumen reduction \( \geq 50\% \) or known coronary heart disease. Black squares indicate the adjusted odds ratios (OR) computed in all study patients (N=378), and gray squares indicate the adjusted OR computed in patients with no known coronary heart disease (N=315). Model 1, Logistic regression analysis adjusted for age and gender. Model 2, Logistic regression analysis adjusted for age, gender, hypertension, dyslipidemia, current smoking, and family history of coronary heart disease.
Coronary Artery Disease and Severity of Extracardiac Atherosclerosis

We found a marked increase in the prevalence of coronary plaque (especially in those with arterial lumen reduction of ≥50%) with increasing severity of carotid atherosclerosis (Figure 5). Using patients without carotid plaque as the reference group, the age- and gender-adjusted OR for the presence of carotid plaque were 2.10 (95% CI, 1.23–3.59) for carotid stenoses <50% and 5.88 (95% CI, 2.38–14.50) for carotid stenoses ≥50%. This relationship was not modified after additional adjustment for risk factors, with an OR of 1.95 (95% CI, 1.09–3.50) for carotid stenosis <50% and 4.52 (95% CI, 1.78–11.51) for stenosis ≥50%. When only coronary stenoses ≥50% were considered, consistent trends were observed (adjusted OR for trend <0.001). An analysis restricted to patients with no known coronary heart disease yielded similar results (adjusted OR for trend <0.005 for both coronary plaque and coronary stenosis ≥50%).

After categorization of atherosclerosis in the aortic arch into 3 groups (absence, plaque <4 mm, and plaque ≥4 mm), the presence of aortic arch plaque ≥4 mm (but not that of aortic arch plaque <4 mm) was associated with the presence of coronary plaque: fully adjusted OR 2.77 (95% CI, 1.01–7.58) for the presence of aortic arch plaque ≥4 mm and 1.02 (95% CI, 0.53–1.94) for presence of aortic arch plaque <4 mm. However, we found no association of coronary stenosis ≥50% with aortic arch plaque ≥4 mm (adjusted OR, 1.34; 95% CI, 0.62–2.90) and with aortic arch plaque <4 mm (OR, 0.61; 95% CI, 0.31–1.20). Similar results were found when analyses were restricted to patients with no known coronary heart disease (data not shown).

Discussion

Based on systematic coronary angiography in a large series of consecutive patients with a nonfatal cerebral infarction and no known coronary heart disease, this study documents a high prevalence of coronary atherosclerosis. Previous studies were based on exercise electrocardiograms or myocardial scintigraphy in patients with stroke or transient ischemic attack.11–22 These small series suggested a prevalence of silent myocardial ischemia as high as 40%. In the present study, which, to our knowledge, is the largest ever using gold standard coronary angiography in nonfatal ischemic strokes, the 62% prevalence of coronary artery plaque and the 26% prevalence of coronary artery stenosis ≥50% was consistent with the 70% prevalence and 29% prevalence, respectively, found in a previous autopsy series of fatal stroke without coronary heart disease history.23 Intravascular ultrasound would have been more sensitive than angiography but was not considered feasible to maintain the consecutive inclusion of patients in this study. Although cardiac CT angiography has been found to be as effective as conventional coronary angiography in detecting coronary stenosis ≥50%, radiation exposure is significantly greater and it is not sufficiently accurate to warrant replacing coronary angiography.23

The high burden of coronary artery disease in patients with cerebral infarction and no known coronary heart disease points to the importance of secondary prevention of coronary heart disease. It has been shown recently that routine revascularization does not improve major clinical outcomes compared with intensive medical therapy in stable coronary heart disease, but it improves symptom control.24,25 Therefore, despite our findings, there will be no need to systematically image coronary arteries in patients with cerebral infarction, unless patients are symptomatic or have evidence of ischemia on noninvasive tests.

In past years, stroke has been advocated as the preferred primary end point in secondary stroke prevention trials of antiplatelet agents, arguing that stroke is the most common event after a stroke.26 Our study strengthens the case that randomized, controlled trials evaluating drugs that control risk factors or platelet inhibition should focus not only on stroke events but also on the sample size, which should be powered to detect an effect on major coronary events.27 Contrary to short-term (1.5–3 years) antiplatelet trials that found a significant reduction in the stroke end point and no significant effect on major coronary events,28–32 long-term (4–5 years) blood pressure or lipid-lowering trials have
shown large reductions in rates of major coronary events in patients with stroke.33,34 This was exemplified in the SPARCL trial, which had included only stroke patients with no known coronary heart disease.34

We could also assess the relationships between systemic atherosclerosis burden and that of coronary atherosclerosis. We found a significant association between the presence of coronary artery disease and carotid and femoral atherosclerosis in the overall group, including patients with either known or no known history of coronary heart disease. Analyses restricted to the group with silent coronary atherosclerosis (ie, no known coronary heart disease) found the same trends. Multiple territory disease has been shown to predict higher global risk in the REACH registry.35 In the present study, having both femoral and carotid artery plaques was a good predictor of the presence of coronary artery disease (Figure 5), with a positive predictive value of 84% for coronary artery plaque. However, there was also a poor negative predictive value for coronary artery plaque of 44%, indicating that even in the absence of systemic atherosclerotic disease there was a high burden of coronary plaques. We could not confirm that aortic arch plaques ≥4 mm also predicted the presence of coronary artery disease (Figure 3), as we and others36–39 have previously shown that they predict stroke and recurrent vascular events.

This study has limitations and strengths. It was limited by the lack of an age- and gender-matched control group. However, collecting a large sample of controls with coronary angiography would have been technically difficult; our pre-

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\[\text{Disclosure}\]

None.

\[\text{References}\]


\[\text{Acknowledgments}\]

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뇌경색 환자에서 관심동맥경화증의 유병률

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(Stroke. 2011;42:22-29.)

Key Words: atherosclerosis ■ cerebral infarction ■ coronary arteries ■ myocardial infarction
Figure 1. Prevalence of asymptomatic coronary artery disease by number of diseased vessels among 315 patients with no history of coronary heart disease. *Regardless of stenosis severity. †Plaque with arterial lumen reduction ≥50% in diameter.

Figure 5. Distribution of coronary artery disease subgroups according to severity of carotid atherosclerosis. CHD, coronary heart disease.