Risk Factors Associated With Severity and Location of Intracranial Arterial Stenosis

Tanya N. Turan, MD; Achraf A. Makki, MD; Samuel Tsappidi, MD; George Cotsonis, MA; Michael J. Lynn, MS; Harry J. Cloft, MD, PhD; Marc I. Chimowitz, MBChB; for the WASID Investigators

Background and Purpose—We sought to determine the vascular risk factors and demographic features associated with the severity and location of intracranial stenosis.

Methods—Data on patients enrolled in the Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial were used for the analyses. Demographic features and vascular risk factors were compared in patients with moderate stenosis (n = 336) versus severe stenosis (n = 225) and according to the location of intracranial stenosis (middle cerebral, internal carotid, basilar, or vertebral artery).

Results—History of a lipid disorder (77% in severe vs 67% in moderate, P = 0.01), metabolic syndrome (63% in severe vs 53% in moderate, P = 0.05), and diabetes (43% in severe vs 35% in moderate, P = 0.04) were more common in patients with severe intracranial stenosis by univariate analyses. A history of a lipid disorder was independently associated with severe stenosis (odds ratio = 1.62; 95% CI, 1.09 to 2.42; P = 0.02). The distribution of stenosis location differed among age groups (P = 0.0015), sexes (P = 0.0001), races (P = 0.0243), qualifying events (P = 0.0156), diabetes (P = 0.0030), coronary artery disease (P = 0.0030), and hyperlipidemia (P = 0.054). Patients with basilar artery stenoses were older and more likely to have hyperlipidemia. Patients with middle cerebral artery stenoses were more likely to be women and black. Patients with internal carotid artery stenoses were more likely to have diabetes. Patients with vertebral artery stenoses were more likely to have coronary artery disease.

Conclusions—History of a lipid disorder had the strongest association with severity of intracranial stenosis and should be the target of prevention therapies. Different locations of intracranial stenoses are associated with different vascular risk factors and demographic features, suggesting that there may be a difference in the underlying pathophysiology of stenoses among the intracranial arteries.

Key Words: intracranial stenosis ■ risk factors ■ cerebral arteries ■ cerebrovascular disease

Atherosclerotic stenosis of the major intracranial arteries (carotid siphon, middle cerebral artery [MCA], vertebral artery, and basilar artery) may be the most common cause of stroke worldwide and is responsible for ≈8% to 10% of all ischemic strokes in the United States. Patients with symptomatic severe (70% to 99%) intracranial stenoses have been shown to have a risk as high as 25% in 2 years for recurrent stroke. Certain risk factors, such as lipoprotein(a) and diabetes, have been associated with the number of intracranial atherosclerotic stenoses, but no large cohort study has examined the risk factors associated with severe (70% to 99%) intracranial arterial stenosis. Identification of risk factors associated with severe stenosis could lead to new strategies for preventing the progression of mild or moderate stenosis to severe intracranial stenosis.

Although identifying risk factors for severe intracranial stenosis may be useful for treatment recommendations, identifying risk factors associated with the location of intracranial stenosis may be important for understanding the pathogenesis of intracranial plaque. In this regard, other studies have evaluated the relationship between risk factors and location of atherosclerotic intracranial stenosis, but those studies have largely been small, single-center, retrospective studies. Completion of the Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) trial has provided a unique opportunity for analysis of the relationship between risk factors, and location and severity of intracranial stenosis in a larger cohort of patients enrolled in a multicenter, prospective, clinical trial.

Methods

The WASID Study enrolled 569 patients with symptomatic intracranial stenosis. For the analysis of risk factors and severity of stenosis, the number of patients analyzed with each risk factor was <569 because of missing baseline risk factor data for some
patients (Table 1). Similarly, for the analysis of risk factors and location of stenoses, the number of patients analyzed with each risk factor was <569 because 34 patients were excluded due to multiple arteries with stenoses and others had missing data on the location of stenosis and baseline risk factors (Table 2).

Details of the WASID study design have been published previously. In brief, inclusion criteria included transient ischemic attack or nondisabling stroke that occurred within 90 days before randomization that was attributable to angiographically proven 50% to 99% stenosis of a major intracranial artery (internal carotid [ICA], MCA, vertebral artery, or basilar artery). Exclusion criteria included tandem 50% to 99% stenosis of the extracranial carotid artery, nonatherosclerotic stenosis of an intracranial artery (intracranial or extracranial dissection, moyamoya disease, vasculitis, radiation-induced vasculopathy, or fibromuscular dysplasia), a cardiac source of embolism, and a contraindication to aspirin or warfarin therapy.

Patients were enrolled on the basis of a local reading of their angiogram. However, final determination of the degree and location of stenosis was based on the WASID study neurologist who used the WASID technique to eliminate interexaminer variability. The analysis was performed by a central neuroradiologist who used the WASID technique to determine the degree of stenosis and severity of stenosis. Severe stenosis was defined as a single lesion with ≥70% diameter stenosis or multiple intracranial lesions (including asymptomatic stenoses) with ≥50% diameter stenosis.

Vascular risk factors were determined at study entry by the WASID study neurologist who used the following definitions. (1) History of a lipid disorder was deemed present when the patient had been treated with either diet therapy or lipid-lowering medications or when the patient met any of the following criteria: total cholesterol >240 mg/dL, LDL >130 mg/dL, HDL <35 mg/dL (men) or 44 mg/dL (women), or triglycerides >250 mg/dL. Lipid levels were measured within 90 days before enrollment, or if this condition had not been met, these measurements had to have been taken within 48 hours of the qualifying event or between 6 weeks and 4 months after the qualifying event, because cholesterol levels may decline after acute stroke. (2) History of hypertension was deemed present when the patient had been treated with antihypertensive agents or had a blood pressure >150 mm Hg systolic or >90 mm Hg diastolic on at least 2 occasions during a 3-month period. (3) History of diabetes was deemed present when the patient had been treated with hypoglycemic agents or had an elevated fasting venous plasma glucose level >125 mg/dL on at least 2 occasions. (4) History of coronary artery disease was deemed present when the patient had a history of myocardial infarction, angina, coronary angioplasty, or coronary bypass surgery.

For the severity of stenosis analyses, the following demographic variables and vascular risk factors at study entry were compared between patients with and without severe stenosis by univariate and multivariate analyses: sex, race (black, white, or other), hypertension, tobacco use, diabetes mellitus, lipid disorder, coronary artery disease, history of previous ischemic stroke, and metabolic syndrome (according to the WASID classification for metabolic syndrome described previously). Demographic variables and vascular risk factors at study entry were compared between patients with and without severe stenosis by a χ² test. Logistic regression was used to assess multivariate significance and to investigate the interaction between diabetes and lipid disorder.

For the location of stenosis analyses, the proportion of the following demographic variables and vascular risk factors at study entry were compared between patients with 50% to 99% stenosis of the intracranial ICA, MCAs, vertebreal arteries, and basilar arteries: age (<64 years or ≥64 years), sex (black, white, or other), diabetes mellitus, lipid disorder, coronary artery disease, and type of qualifying event (stroke or transient ischemic attack). Exact binomial methods were used to calculate 95% CIs for these proportions. These variables were also compared between an anterior location (ICA or MCA) versus a posterior location (vertebral or basilar) of stenosis. To assess differences in risk factors among the 4 locations (ICA, MCA, vertebral, basilar), log-linear methods were used.

### Table 1. Comparison of Vascular Risk Factors at Study Entry Between Patients With Severe and Moderate Stenoses

<table>
<thead>
<tr>
<th>Vascular Risk Factor</th>
<th>Degree of Stenosis</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Severe Stenosis, % (No. of Patients)</td>
<td>Moderate Stenosis, % (No. of Patients)</td>
</tr>
<tr>
<td></td>
<td>With Stenosis/No. With Stenosis</td>
<td>With Risk Factor/No. With Moderate Stenosis</td>
</tr>
<tr>
<td>Hypertension</td>
<td>83% (187/225)</td>
<td>85% (283/334)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>43% (97/224)</td>
<td>35% (117/336)</td>
</tr>
<tr>
<td>Lipid disorder</td>
<td>77% (166/216)</td>
<td>67% (220/328)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>26% (58/220)</td>
<td>28% (92/330)</td>
</tr>
<tr>
<td>Previous ischemic stroke</td>
<td>24% (54/221)</td>
<td>25% (81/328)</td>
</tr>
<tr>
<td>Smoking</td>
<td>67% (150/225)</td>
<td>63% (211/336)</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>63% (112/179)</td>
<td>53% (159/302)</td>
</tr>
</tbody>
</table>

*Significant difference between groups.

### Table 2. Percentages of Patients With Risk Factor or Demographic Characteristic by Location of Intracranial Stenosis

<table>
<thead>
<tr>
<th>Location of Stenosis</th>
<th>ICA (n=119), % (95% CI)*</th>
<th>MCA (n=179), % (95% CI)*</th>
<th>Vertebral (n=107), % (95% CI)*</th>
<th>Basilar (n=112), % (95% CI)*</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;64 y</td>
<td>42 (33,51)</td>
<td>42 (35,49)</td>
<td>51 (42,61)</td>
<td>63 (54,72)</td>
<td>0.0015</td>
</tr>
<tr>
<td>Female</td>
<td>34 (26,44)</td>
<td>50 (43,58)</td>
<td>25 (17,35)</td>
<td>33 (24,43)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Black‡</td>
<td>30 (22,39)</td>
<td>40 (32,47)</td>
<td>27 (19,37)</td>
<td>24 (17,33)</td>
<td>0.0243</td>
</tr>
<tr>
<td>Stroke as qualifying event§</td>
<td>63 (54,72)</td>
<td>70 (63,77)</td>
<td>56 (46,66)</td>
<td>54 (44,63)</td>
<td>0.0156</td>
</tr>
<tr>
<td>Diabetes</td>
<td>50 (41,60)</td>
<td>33 (26,40)</td>
<td>41 (32,51)</td>
<td>29 (21,39)</td>
<td>0.0030</td>
</tr>
<tr>
<td>Hypertension</td>
<td>85 (77,91)</td>
<td>83 (76,88)</td>
<td>85 (77,91)</td>
<td>88 (81,94)</td>
<td>0.6130</td>
</tr>
<tr>
<td>Lipid disorder</td>
<td>73 (64,81)</td>
<td>64 (57,71)</td>
<td>73 (63,81)</td>
<td>79 (70,86)</td>
<td>0.0540</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>32 (24,41)</td>
<td>19 (13,25)</td>
<td>38 (29,49)</td>
<td>27 (19,36)</td>
<td>0.0030</td>
</tr>
</tbody>
</table>

*Percentage (95% CI) of patients with the risk factor or demographic characteristic who had stenosis in the location of the artery of interest.
†χ² P value comparing the proportion of risk factor between the location of stenosis.
‡Compared with white or other race.
§Compared with transient ischemic attack.
MCA, vertebral, or basilar artery), a $\chi^2$ test followed by Tukey’s post hoc comparisons was used to test the location of stenosis versus demographic variables and vascular risk factors at study entry. Probability values $<0.05$ were considered significant.

Results

Risk Factors for Severe Stenosis

Of the 561 patients analyzed, 225 (40%) had severe stenosis. The univariate analysis of risk factors at study entry associated with severe intracranial stenosis is represented in Table 1. Diabetes, lipid disorder, and metabolic syndrome were significantly more common in patients with severe stenosis than in patients with moderate stenosis. There was no significant difference in the percentage of males and females with severe stenosis (41% vs 39%, $P=0.64$). However, there was a trend toward a lower frequency of severe stenosis in blacks (34% of blacks, 42% of whites, 48% of other race; $P=0.06$). In the multivariate analysis, history of a lipid disorder was the only independent predictor of severe intracranial stenosis (odds ratio $=1.62$; 95% CI, 1.09 to 2.42; $P=0.02$).

Because both diabetes and a lipid disorder were predictors of severe stenosis in the univariate analyses, we checked for a possible synergistic interaction between these 2 vascular risk factors. The percentage of patients with neither risk factor who had severe stenosis was 29%, those with only a lipid disorder who had severe stenosis was 40%, those with only diabetes who had severe stenosis was 39%, and those with both risk factors who had severe stenosis was 47%. The probability value for the interaction was 0.6918.

Risk Factors by Location of Stenosis

The distribution of the location of stenoses among the major arteries was as follows: 179 MCA (34.62%), 119 ICA (23.0%), 107 vertebral (20.69%), and 112 basilar (21.66%). The percentage of patients with risk factors for each intracranial arterial location is shown in Table 2. The distribution of stenosis location differed among age groups ($P=0.0015$), sexes ($P=0.0001$), races ($P=0.0243$), qualifying events (0.0156), history of diabetes ($P=0.0030$), and history of coronary artery disease ($P=0.0030$). There was a trend for the distribution of stenosis location to differ according to a history of hyperlipidemia ($P=0.054$). A history of hypertension was equally present in patients with stenoses in all locations.

Pairwise comparison of the percentages of patients with various risk factors who had stenoses in the 4 locations are as follows (only significant associations $P<0.05$ are reported): The percentage of patients with basilar stenosis who were old (age $>64$) (63%) was greater than the percentages of older patients with ICA (42%) and MCA (42%) stenoses. The percentage of patients with MCA stenosis who were women (50%) was greater than the percentages of women patients with ICA (34%), vertebral (25%), or basilar (33%) stenoses. The percentage of patients with MCA stenosis who were black (40%) was greater than the percentage of black patients with basilar (24%) stenosis. The percentage of patients with MCA stenosis who had a stroke as the qualifying event (70%) was greater than the percentage of patients with basilar (54%) stenosis who had stroke as the qualifying event. There was a higher rate of history of diabetes (50%) in patients with an ICA stenosis than with MCA (33%) or basilar (30%) stenoses. There was a higher rate of a history of coronary artery disease (38%) in patients with vertebral stenosis than with MCA (19%) stenosis. There was a higher rate of a history of hyperlipidemia (79%) in patients with basilar stenosis than with MCA (64%) stenosis.

Discussion

Previous studies have shown that symptomatic intracranial stenosis is associated with a higher frequency of hypertension, hypercholesterolemia, tobacco use, diabetes, black race, and male sex compared with other causes of ischemic stroke, but to our knowledge, this is the first study to correlate vascular risk factors with the severity of angiographically proven symptomatic intracranial stenosis.

Identifying the factors associated with severe (70% to 99%) intracranial stenosis is important because patients with severe intracranial stenosis have been shown to have the highest rate of recurrent stroke. Previous analysis of the WASID cohort demonstrated that patients with 70% to 99% intracranial stenosis have a $>2$-fold risk of stroke in the territory of the stenotic artery than do patients with $<70$% stenosis. In this study, we found that history of a lipid disorder was significantly associated with severe stenosis. This suggests the possibility that early and effective treatment of lipid disorders may prevent the progression of intracranial atherosclerosis, thereby decreasing the high risk of recurrent ischemic stroke. Prior analyses of the WASID data have shown that patients with intracranial stenosis who have elevated cholesterol levels are at higher risk of stroke, providing further evidence that lipid lowering should be a primary target in this disease.

A surprising finding in our study was a trend toward a lower percentage of severe stenosis in blacks compared with other racial groups. Previous studies have shown that blacks have a higher incidence of intracranial atherosclerosis overall than do other racial groups, and a previous autopsy report demonstrated that blacks tend to have more raised atherosclerotic lesions of the intracranial arteries than do whites. However, given that the autopsy study was not limited to patients with symptomatic intracranial stenosis and that only symptomatic patients were included in the WASID cohort, the lower percentage of black patients with severe stenosis in this study could suggest that blacks may become symptomatic at a lower degree of stenosis. On the other hand, our finding that blacks may have less severe intracranial stenosis, despite a higher incidence of the disease, may be due to the selected patient study population in WASID. Patients with severe disabling strokes were excluded from the WASID study, and therefore it is possible that blacks with the most severe intracranial stenosis may have had more disabling strokes, which prevented their inclusion in this cohort.

Our findings that different risk factors are associated with different locations of intracranial stenosis are consistent with previous reports. Yasaka et al reported an association between hyperlipidemia and basilar stenosis in Japanese and mixed-race North American populations, respectively. Caplan et al also reported that MCA disease is
more common in black females, and Gorelick et al.\textsuperscript{7} reported that blacks have higher rates of anterior circulation atherosclerosis, also consistent with our findings. Both Yasaka et al.\textsuperscript{8} and Gorelick et al.\textsuperscript{7} also found that hypertension did not predict lesion site, similar to our findings. Our findings that vertebral artery stenosis is associated with coronary artery disease and that intracranial carotid stenosis is related to diabetes have been previously shown in an asymptomatic Japanese population\textsuperscript{9} as well. On the other hand, our finding that basilar stenosis is associated with older age is not consistent with other reports\textsuperscript{9,20} that found no association between age and location of stenosis. However, most of the studies mentioned earlier either did not include patients with stenoses in all 4 major intracranial arterial locations (intracranial ICA, MCA, vertebral, and basilar)\textsuperscript{5-7} or included patients with extracranial carotid\textsuperscript{5,6,8} or vertebral\textsuperscript{20} stenosis and therefore were not specifically comparing differences in risk factors among intracranial stenosis locations. Our findings are unique in that WASID is the largest prospective cohort of patients with angiographically verified symptomatic intracranial stenosis, and therefore, this is the largest comparison of risk factors by location of stenosis in a mixed-race North American population.

The explanation for the association between certain risk factors and location of intracranial stenosis in this and other studies is unclear. One possible explanation is that the interface between systemic factors, such as vascular risk factors or genetic factors, and local factors, such as hemodynamic or structural factors, may differ by arterial location. Further studies of the composition of intracranial atherosclerotic lesions in different intracranial arteries may help to clarify this issue. Emerging noninvasive in vivo technologies, such as high-resolution magnetic resonance imaging, may shed further light on the differences in risk factors and demographic features associated with certain locations of intracranial stenosis.

Interestingly, we found that the qualifying event (stroke or transient ischemic attack) differed by location of stenosis, as the group with basilar stenoses had the highest percentage of patients presenting with transient ischemic attack and the group with MCA stenoses had the highest percentage of patients presenting with stroke. It is possible that this finding is the result of selection bias in WASID; that is, patients with basilar stenosis and stroke may have been thought to have been too high-risk for randomization and were not entered into the study. Another possibility, given that WASID excluded patients with severe disabling stroke, is that patients with basilar stenosis have had more severe strokes, and therefore, fewer basilar stenosis patients with stroke were eligible for WASID.

The main limitation of our study is that it was a post hoc analysis of patients enrolled in a clinical trial rather than a population-based study. Despite this limitation, our findings indicate that history of a lipid disorder has the strongest association with severity of intracranial stenosis and should be the target of prevention therapies. Different locations of intracranial stenosis are associated with different vascular risk factors and demographic features, suggesting that there may be a difference in the underlying pathophysiology of atherosclerotic stenosis between the intracranial arteries. Further studies of intracranial artery structure and mechanisms of atherosclerosis pathogenesis are needed to clarify this issue.

Sources of Funding

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Disclosures

Marc Chimowitz, MBChB, reports being paid fees by the Bristol-Myers Squibb/Sanofi Pharmaceuticals Partnership, Astra-Zeneca, and the Sankyo Lilly Partnership for consulting on antithrombotic agents that were not evaluated in this trial and from Guidant Corporation for consulting on a medical device (an intracranial stent) that was not evaluated in this trial. There are no other conflicts to report.

References

10. Warfarin-Aspirin Symptomatic Intracranial Disease Trial Investigators. Design, progress and challenges of a double-blind trial of warfarin versus
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Abstract

与颅内动脉狭窄的部位及严重程度相关的危险因素

Risk Factors Associated With Severity and Location of Intracranial Arterial Stenosis

Tanya N. Turan, MD; Achraf A. Makki, MD; Samuel Tsappidi, MD; George Cotsonis, MA; Michael J. Lynn, MS; Harry J. Cloft, MD, PhD; Marc I. Chimowitz, MBChB; for the WASID Investigators

背景和目的：探索与颅内动脉狭窄的部位及严重程度相关的血管性危险因素和人口学特点。

方法：对纳入华法林-阿司匹林治疗症状性颅内病变（Warfarin-Aspirin Symptomatic Intracranial Disease, WASID）研究的患者数据进行分析，比较中度血管狭窄患者（n=336）与重度血管狭窄患者（n=225）之间，以及不同部位动脉狭窄的患者（大脑中动脉、颈内动脉、基底动脉或椎动脉）之间，血管性危险因素和人口学特点。

结果：单因素分析显示，重度颅内动脉狭窄患者中，既往有血脂异常史（重度狭窄77%，中度狭窄67%，P=0.01），代谢综合征史（重度狭窄63%，中度狭窄53%，P=0.05）以及糖尿病史（重度狭窄43%，中度狭窄35%，P=0.04）更常见。血脂异常史是重度动脉狭窄的独立危险因素（OR=1.62; 95% CI: 1.09-2.42; P=0.02）。动脉狭窄的部位在不同年龄（P=0.0015）、性别（P=0.0243）以及是否受血管事件（P=0.0156）、糖尿病史（P=0.0030）和高脂血症（P=0.054）等方面存在差异。基底动脉狭窄的患者年龄较大，且患有高脂血症的比例更高，大脑中动脉狭窄在女性及黑人中更为常见。颅内动脉狭窄的患者合并糖尿病的比例较高，椎动脉狭窄的患者合并冠心病的比例更高。

结论：鉴于血脂异常与颅内动脉狭窄程度密切相关，故应成为预防治疗的靶点。不同部位的颅内动脉狭窄与不同的血管性危险因素和人口学特点相关，提示存在不同的病理生理机制。

关键词：颅内动脉狭窄，危险因素，脑动脉，脑血管病

表1 重度和中度颅内动脉狭窄患者的基线血管性危险因素比较

<table>
<thead>
<tr>
<th>血管性危险因素</th>
<th>重度狭窄, % (有危险因素的患者数 / 血管重度狭窄的患者数)</th>
<th>中度狭窄, % (有危险因素的患者数 / 血管中度狭窄的患者数)</th>
<th>P值</th>
</tr>
</thead>
<tbody>
<tr>
<td>高血压</td>
<td>83% (187/225)</td>
<td>85% (283/334)</td>
<td>0.61</td>
</tr>
<tr>
<td>糖尿病</td>
<td>43% (97/224)</td>
<td>35% (117/336)</td>
<td>0.04*</td>
</tr>
<tr>
<td>血脂异常</td>
<td>77% (166/216)</td>
<td>67% (220/328)</td>
<td>0.01*</td>
</tr>
<tr>
<td>冠心病</td>
<td>26% (58/220)</td>
<td>28% (92/330)</td>
<td>0.70</td>
</tr>
<tr>
<td>既往卒中史</td>
<td>24% (54/221)</td>
<td>25% (81/328)</td>
<td>0.94</td>
</tr>
<tr>
<td>吸烟史</td>
<td>67% (150/225)</td>
<td>63% (211/336)</td>
<td>0.35</td>
</tr>
<tr>
<td>代谢综合征</td>
<td>63% (112/179)</td>
<td>53% (159/302)</td>
<td>0.05*</td>
</tr>
</tbody>
</table>

*组间比较存在统计学差异。

表2 不同部位颅内血管狭窄的患者存在危险因素或人口学特点的百分比

<table>
<thead>
<tr>
<th>血管部位</th>
<th>颈内动脉 (n=119)</th>
<th>大脑中动脉 (n=179)</th>
<th>椎动脉 (n=107)</th>
<th>基底动脉 (n=112)</th>
<th>P值</th>
</tr>
</thead>
<tbody>
<tr>
<td>年龄 &gt;64 岁</td>
<td>42(33,51)</td>
<td>42(35,49)</td>
<td>51(42,61)</td>
<td>63(54,72)</td>
<td>0.0015</td>
</tr>
<tr>
<td>女性</td>
<td>34(26,44)</td>
<td>50(43,58)</td>
<td>25(17,35)</td>
<td>33(24,43)</td>
<td>0.001</td>
</tr>
<tr>
<td>手术部人员</td>
<td>30(22,39)</td>
<td>40(32,47)</td>
<td>27(19,37)</td>
<td>24(17,33)</td>
<td>0.0243</td>
</tr>
<tr>
<td>卒中事件 §</td>
<td>63(54,72)</td>
<td>70(63,77)</td>
<td>56(46,66)</td>
<td>54(44,63)</td>
<td>0.0156</td>
</tr>
<tr>
<td>糖尿病</td>
<td>50(41,60)</td>
<td>33(26,40)</td>
<td>41(32,51)</td>
<td>29(21,39)</td>
<td>0.0030</td>
</tr>
<tr>
<td>高血压</td>
<td>85(77,91)</td>
<td>83(76,88)</td>
<td>85(77,91)</td>
<td>88(81,94)</td>
<td>0.6130</td>
</tr>
<tr>
<td>血脂异常</td>
<td>73(64,81)</td>
<td>64(57,71)</td>
<td>73(63,81)</td>
<td>79(70,86)</td>
<td>0.0540</td>
</tr>
<tr>
<td>冠心病</td>
<td>32(24,41)</td>
<td>19(13,25)</td>
<td>38(29,49)</td>
<td>27(19,36)</td>
<td>0.0030</td>
</tr>
</tbody>
</table>

*在相应动脉发生狭窄的患者中存在危险因素或人口学特点的百分比 (95% CI)。
†P值比较不同狭窄部位的危险因素比率。
‡与白人或其他人种比较。
§与发生 TIA 患者的比较。