Prevalence and Calcification of Intracranial Arterial Stenotic Lesions as Assessed With Multidetector Computed Tomography Angiography

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Background and Purpose—Intracranial arterial stenosis (ICAS) in patients with recent ischemic stroke is associated with a high risk of recurrent stroke. More insight into the pathophysiology of ICAS could help identify patients at high risk requiring more aggressive secondary prevention. We evaluated the prevalence, distribution, calcification, and the risk factors predisposing ICAS in a European stroke population.

Methods—Consecutive patients with a transient ischemic attack or ischemic stroke (n=786) were evaluated for the presence and distribution of ICAS (≥30% luminal narrowing) by CT angiography. ICAS were categorized as symptomatic or asymptomatic, and the presence of calcification was assessed. The association of traditional cerebrovascular risk factors and the erythrocyte sedimentation rate with ICAS was analyzed.

Results—In 178 of 786 patients (23%), 288 ICAS were observed. Most stenoses (n=194/288; 67%) were located in the posterior circulation arteries. In 59 of 786 patients (8%), ICAS were considered symptomatic. ICAS in the basilar artery and arteries beyond the circle of Willis were mainly noncalcified. In addition to age, gender, and several traditional cerebrovascular risk factors, erythrocyte sedimentation rate was independently associated with the presence of ICAS (OR, 1.20; 95% CI, 1.06–1.36) and with the presence of noncalcified ICAS in particular (OR, 1.20; 95% CI, 1.05–1.37).

Conclusions—ICAS was observed in a noteworthy number of European stroke patients. Particularly, the majority of ICAS was observed in the posterior circulation, possibly conferring worse prognosis. ICAS in distal arteries were mainly noncalcified. Association of noncalcified ICAS and erythrocyte sedimentation rate may indicate a prominent role for inflammatory factors in intracranial atherosclerotic disease. **(*Stroke*. 2011;42:1244-1250.)**

Key Words: atherosclerosis ■ atherosclerotic plaque calcification ■ computed tomography ■ intracranial stenosis ■ risk factors

Intracranial arterial stenosis (ICAS) in patients with TIA or ischemic stroke is associated with a high risk of recurrent stroke.[1] Angioplasty and stenting are feasible procedures for revascularization of vessels affected by ICAS. However, insufficient evidence is available to recommend these treatments for the prevention of recurrent stroke in patients with ICAS in clinical practice.[2] More insight into the prevalence, distribution, and calcification of ICAS lesions could help identify patients at high risk requiring more aggressive secondary prevention.

The prevalence of ICAS seems to vary among ethnic groups.[3] Nevertheless, only limited studies have assessed the prevalence and associated risk factors for ICAS in European stroke patients.[4–6] Moreover, the comparative value of studies available in European patients is limited by the use of multiple imaging modalities.

Also, little is known about the composition of ICAS lesions, which may point to a specific pathophysiological process.[7] The pathophysiology of intracranial atherosclerosis is suggested to differ from that of the extracranial arteries.[8] A prominent role for inflammatory factors is indicated in the atherosclerosis of the intracranial arteries.[9] Consequently, the proatherogenic influence of inflammatory reactions could be manifested as an association between the erythrocyte sedimentation rate (ESR) and ICAS, as previously observed in a single study.[10] In addition, an accelerated intracranial atherogenesis could be reflected in differences in plaque calcification.

Multidetector computed tomography angiography (MDCTA) is reliable for the evaluation of both extracranial[11] and intracranial atherosclerotic disease.[12] Moreover, the technique is available for detection of ICAS in most European

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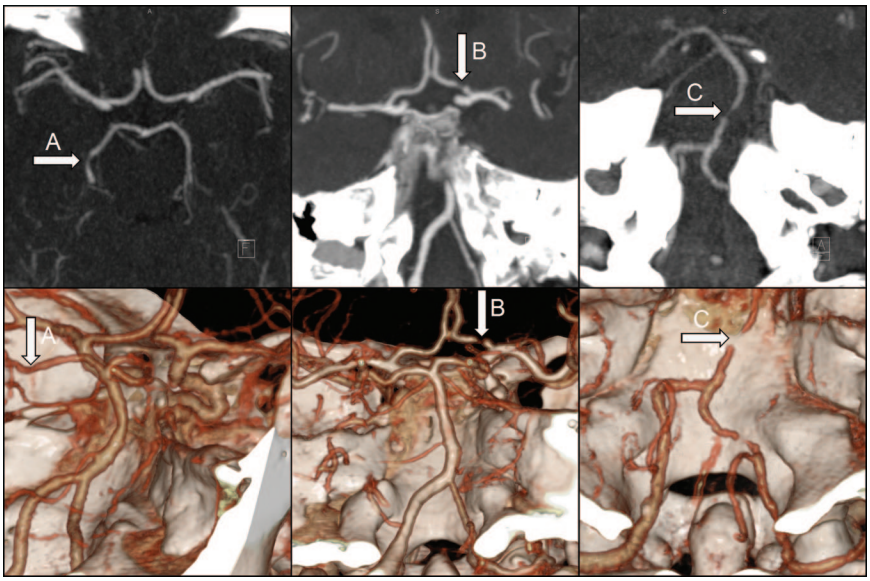

Figure 1. Assessment of intracranial arterial stenosis (ICAS) using multidector CTA. Upper row, maximum intensity projection images of 4-mm thickness demonstrating ICAS 30% to 49% in the right posterior cerebral artery **(A),** ICAS 50% to 69% in the left anterior cerebral artery **(B),** and ICAS 70% to 99% in the basilar artery **(C)**. Lower row, volume-rendering images demonstrating the same ICAS lesions in 3-dimensional anatomy.

hospitals.[13] As compared to digital subtraction angiography, MDCTA has been demonstrated to be effective in the detection of ICAS, with a sensitivity of 97% and a specificity of 99%.[12] In addition, MDCTA allows differentiation between calcified and noncalcified atherosclerotic plaques.[14]

In the current study, we evaluated a large cohort of patients with TIA or ischemic stroke for the prevalence, distribution, and the calcification of ICAS lesions using MDCTA. Furthermore, the association of ICAS with the traditional risk factors for cerebrovascular disease as well as with ESR was investigated.

Materials and Methods

Study Population

From a prospective registry of 911 consenting patients with amaurosis fugax, TIA, or ischemic stroke (Rankin score <4 at discharge), we selected all patients (n=795) with a recent ischemic stroke or TIA but excluded patients with amaurosis fugax. Patients were enrolled from a specialized TIA/stroke outpatient clinic or the stroke unit. All patients were interviewed and examined by a vascular neurologist and underwent electrocardiography and laboratory analysis. Medical history and cerebrovascular risk factors were recorded. On admission, patients underwent MDCT of the brain and MDCTA of the carotid and intracranial arteries in a single session. Three patients with MDCTA of insufficient quality for reliable evaluation and 6 patients with intracranial arteries outside the scan reconstruction area were excluded. Consequently, analyses were performed in the remaining 786 patients.

Risk Factors

Ethnicity of patients was determined through an algorithm based on place of birth of the patients and their parents, as well as on name and surname.[15] For the purpose of this study, we distinguished between Asian and non-Asian ethnicities. History of ischemic heart disease was defined as previous chronic heart failure, angina pectoris, myocardial infarction, or coronary artery bypass grafting. Hypercholesterolemia was defined as fasting cholesterol >5.0 mmol/L or treatment with cholesterol-lowering medication. Hypertension was defined as a systolic blood pressure >140 mm Hg and/or a diastolic blood pressure >90 mm Hg during 2 episodes of at least 15 minutes of continuous noninvasive blood pressure measurement or treatment with antihypertensive medication. Diabetes was defined as fasting serum a glucose level >7.9 mmol/L, HbA1c >6.5%, or use of antidiabetic medication. Laboratory analysis included total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, and glucose, as well as ESR.

MDCT and MDCTA Data Acquisition and Analysis

MDCTA was performed with a 16-slice MDCT scanner (Sensation 16; Siemens) or a 64-slice MDCT scanner (Sensation 64; Siemens) with a standardized protocol.[16,17] Intracranial arteries were evaluated on a stand-alone workstation (Leonardo; Siemens Medical Solutions) with multiplanar reformatting and maximum intensity projection images of 4-mm thickness (Figure 1). Because symptomatic ulceration with superimposed thrombus of intracranial atherosclerotic plaques is also present in low-grade stenosis, we defined ICAS as ≥30% luminal narrowing.[18] The degree of stenosis was measured according to the WASID criteria on oblique multiplanar reformatting images perpendicular to the central lumen line.[19] Stenoses were classified as 30% to 49%, 50% to 69%, and 70% to 99%. The internal carotid arteries, the anterior cerebral arteries, the medial cerebral arteries, the vertebral arteries, the basilar artery, and the posterior cerebral arteries were analyzed.

Blinded to clinical information, 2 trained observers (P.J.H. and G.J.J.P.) independently analyzed the presence of ICAS according the WASID method in the first 50 patients. After 4 weeks, the first observer analyzed the same 50 patients. Good interobserver agreement (κ=0.79; 95% CI, 0.55–1.02) and intraobserver agreement (κ=0.79; 95% CI, 0.60–0.99) were observed.

A calcified ICAS lesion was defined as any intracranial stenosis (≥30%) containing plaque calcifications (>130 Hounsfield units). Symptomatic ICAS was defined as any intracranial stenosis (≥30%) in an artery supplying the involved region of the brain, taking into account the configuration of the circle of Willis.

Statistical Analysis

Differences between variables were tested with the χ^2 test, Fisher exact test, Mann-Whitney *U* test, or a nonparametric rank test when appropriate. The association of traditional cerebrovascular risk factors and ESR with the presence of ICAS was determined using regression analysis. The risk factors significantly associated with ICAS ($P<0.05$) in the univariable regression analysis, which were not directly interrelated, were included in a multivariable regression model. Associations were expressed as OR with 95% CI. The analysis was repeated for the presence of noncalcified ICAS lesions, calcified ICAS lesions, and symptomatic ICAS in patients. For the

Table 1. Baseline Characteristics of the Study Population

	No ICAS (n=608; 77%)	ICAS (n=178; 23%)	P
Age	60±14	68±12	<0.001
Male	332 (55%)	110 (62%)	0.09
Asian	20 (3%)	16 (9%)	0.001
Index event			
TIA	238 (39%)	47 (26%)	0.002
Ischemic stroke	370 (61%)	131 (74%)	
Time since onset (days)	5 (1–14)	5 (0–18)	0.40
Cerebrovascular history			
Previous ischemic stroke	77 (13%)	32 (18%)	0.07
Previous TIA >6 mo	85 (14%)	30 (17%)	0.34
History of ischemic heart disease	101 (17%)	33 (19%)	0.55
Cerebrovascular risk factors			
Hypercholesterolemia	440 (72%)	135 (76%)	0.36
Hypertension	389 (64%)	146 (82%)	<0.001
Diabetes mellitus	92 (15%)	53 (30%)	<0.001
Atrial fibrillation	39 (6%)	14 (8%)	0.50
Smoking	230 (38%)	56 (31%)	0.12
Laboratory results			
Cholesterol (mmol/L)	5.2±1.1	5.3±1.2	0.38
HDL cholesterol (mmol/L)	1.41±0.51	1.40±0.74	0.51
LDL cholesterol (mmol/L)	3.29±1.04	3.47±1.19	0.15
Triglycerides (mmol/L)	1.72±2.54	1.71±0.96	0.16
Glucose (mmol/L)	4.9±1.7	5.7±2.4	<0.001
ESR (mm/hr)	13±12	16±15	0.001
Extracranial carotid artery stenosis ≥50%	50 (8%)	38 (21%)	<0.001

Data are means±SD, median (interquartile range), or number of patients (%).

ESR indicates erythrocyte sedimentation rate; HDL, high-density lipoprotein; ICAS, intracranial arterial stenosis; LDL, low-density lipoprotein; TIA, transient ischemic attack.

purpose of this analysis, patients with solely noncalcified ICAS lesions were considered as "patients with noncalcified ICAS lesions," whereas patients with any calcified ICAS lesions were considered as "patients with calcified ICAS lesions." Statistical analyses were performed using SPSS 15.0. $P<0.05$ was considered statistically significant.

Results

Prevalence, Distribution, and Calcification of ICAS

Most patients were male (n=513; 56%) and the mean age was 62±14 years. Baseline characteristics of patients with and without ICAS are illustrated in Table 1. The presence and severity of ICAS in different arteries are shown in Table 2. ICAS ≥30% was observed in 178 patients (23%). ICAS ≥50% was present in 77 patients (10%), and ICAS ≥70% was present in 21 patients (3%). In total, 288 ICAS (≥30%) were observed.

In 184 of 288 ICAS (64%), the degree of stenosis ranged from 30% to 49%, from 50% to 69% in 83 of 288 ICAS (29%), and from 70% to 99% in the remaining 21 of 288 ICAS (7%). Occlusions were present in 52 arteries. Interestingly, the majority of ICAS (n=194/288; 67%) was located in the posterior circulation. Stenoses ≥70% occurred mainly in the posterior circulation of the brain. Exclusively noncalcified ICAS lesions were observed in 126 patients (16%). In total 221 of 288 ICAS, lesions (77%) were noncalcified. ICAS lesions in the anterior cerebral artery, medial cerebral artery, and basilar artery were exclusively noncalcified. Calcified ICAS lesions were predominantly present in the proximal arteries (internal carotid artery and vertebral artery; n=64), whereas only 3 calcified ICAS lesions were identified in the posterior cerebral artery. In 59 patients (8%), a total of 63 symptomatic ICAS ≥30% was observed. Symptomatic ICAS of ≥50% was present in 18 of the patients (3%). Overall, symptomatic ICAS comprised 39 stenoses in the anterior circulation and 24 stenoses in the posterior circulation.

Risk Factors Associated With ICAS

Multivariable analysis revealed an independent association between ICAS and age, male gender, Asian ethnicity, hypertension, diabetes mellitus, LDL cholesterol, and ESR (Table 3). Risk factors independently associated with noncalcified ICAS lesions and calcified ICAS lesions are provided in Table 4. Age, male gender, hypertension, diabetes mellitus,

Table 2. Distribution and Severity of Intracranial Arterial Stenosis (n=288)

		Degree of Stenosis					
Artery	No. of ICAS	30–49%	50–69%	70–99%	Occlusions	Calcified ICAS Lesions	Symptomatic ICAS
Anterior (n=94; 33%)							
ICA	31 (11%)	24 (8%)	7 (2%)	0 (0%)	14	25 (9%)	15 (5%)
ACA	17 (6%)	11 (4%)	6 (2%)	0 (0%)	0	0 (0%)	4 (1%)
MCA	46 (16%)	30 (10%)	14 (5%)	2 (1%)	20	0 (0%)	20 (7%)
Posterior (n=194; 67%)							
VA	88 (31%)	57 (20%)	24 (8%)	7 (2%)	4	39 (14%)	8 (3%)
BA	30 (10%)	16 (6%)	8 (3%)	6 (2%)	5	0 (0%)	8 (3%)
PCA	76 (26%)	46 (16%)	24 (8%)	6 (2%)	9	3 (1%)	8 (3%)
Total	288 (100%)	184 (64%)	83 (29%)	21 (7%)		67 (23%)	63 (22%)

ACA indicates anterior cerebral artery; BA, basilar artery; ICA, internal carotid aretery; ICAS, intracranial arterial stenosis; MCA, medial cerebral artery; PCA, posterior cerebral artery; VA, vertebral artery.

Data are No. of ICAS (% of all ICAS).

Table 3. Univariable and Multivariable Analysis of Risk Factors Associated With Intracranial Arterial Stenosis

	OR (95% CI)	OR (95% CI)
Age (per 10 y)*	1.65 (1.43–1.90)	1.65 (1.40–1.94)
Male	1.34 (0.96–1.89)	1.55 (1.05–2.29)
Asian	2.90 (1.47–5.73)	2.68 (1.20–5.98)
Index event		
Ischemic stroke	1.79 (1.24–2.60)	. . .
Cerebrovascular history		
Previous ischemic stroke	1.51 (0.96–2.37)	. . .
Previous TIA >6 mo	1.25 (0.79–1.96)	. . .
History of ischemic heart disease	1.14 (0.74–1.76)	. . .
Cerebrovascular risk factors		
Hypercholesterolemia	1.20 (0.81–1.76)	. . .
Hypertension	2.57 (1.69–3.90)	1.87 (1.16–3.00)
Diabetes mellitus	2.38 (1.61–3.51)	2.08 (1.34–3.22)
Atrial fibrillation	1.25 (0.66–2.35)	. . .
Smoking	0.75 (0.53–1.08)	. . .
Laboratory results		
Cholesterol (mmol/L)*	1.13 (0.97–1.30)	. . .
HDL cholesterol (mmol/L)*	0.99 (0.74–1.34)	. . .
LDL cholesterol (mmol/L)*	1.17 (0.99–1.36)	1.33 (1.12–1.59)
Triglycerides (mmol/L)*	1.00 (0.93–1.08)	. . .
Glucose (mmol/L)*	1.19 (1.10–1.29)	. . .
ESR (per 10 mm/h)*	1.22 (1.09–1.37)	1.20 (1.06–1.36)
Extracranial carotid artery stenosis ≥50%	3.03 (1.91–4.80)	. . .

CI indicates confidence interval; ESR, erythrocyte sedimentation rate; HDL, high-density lipoprotein; ICAS, intracranial arterial stenosis; LDL, low-density lipoprotein; OR, odds ratio; TIA, transient ischemic attack.

*Ratio per unit increase.

LDL cholesterol, and ESR remained independently associated with noncalcified ICAS lesions, whereas only age was independently associated with calcified ICAS lesions. The median time since symptom onset and clinical and laboratory analysis was 5 days.[1–14] The association of ESR with ICAS remained present after adjustment for time between onset of symptoms (OR, 1.22; 95% CI, 1.08–1.36). Interestingly, ESR increased with degree of intracranial stenosis (Figure 2). Age, Asian ethnicity, hypertension, and ESR were independently associated with symptomatic ICAS.

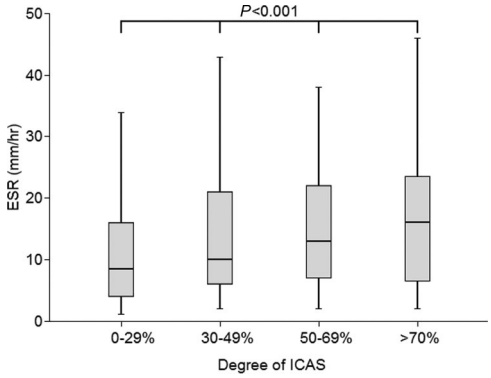

Figure 2. Box plot illustrating higher median erythrocyte sedimentation rates with increase of severity of stenosis.

Discussion

In the current study, ICAS ≥30% was observed in 178 patients (23%) with a recent ischemic stroke or TIA. The majority of all ICAS was located in the posterior circulation arteries. Symptomatic ICAS ≥30% was observed in 59 patients (8%). Calcified ICAS lesions were predominantly observed in the proximal intracranial arteries (internal carotid artery and vertebral artery), whereas ICAS lesions in the distal intracranial arteries (basilar artery, anterior cerebral artery, medial cerebral artery, and posterior cerebral artery) were mainly noncalcified. A number of traditional risk factors including age, male gender, Asian ethnicity, hypertension, diabetes mellitus, and LDL cholesterol were independently associated with the presence of ICAS in multivariable analysis. An independent association was also observed between ESR and ICAS. Equivalent traditional risk factors and ESR were also associated with noncalcified ICAS lesions. However, age was the only risk factor associated with calcified ICAS lesions.

Prevalence and Distribution of ICAS

The comparability of studies on the prevalence of ICAS in stroke patients is limited because of the variation in the

Table 4. Multivariable Analysis of Risk Factors Associated With Exclusively Noncalcified Intracranial Arterial Stenosis Lesions, Calcified Intracranial Arterial Stenosis Lesions, and Symptomatic Intracranial Arterial Stenosis

	OR (95% CI) Noncalcified ICAS Lesions (n=126; 16%)	OR (95% CI) Calcified ICAS Lesions (n=52; 7%)	OR (95% CI) Symptomatic ICAS (n=59; 8%)
Age (per 10 y)*	1.37 (1.15–1.63)	2.09 (1.56–2.82)	1.37 (1.07–1.75)
Male	1.56 (1.01–2.40)	1.29 (0.68–2.45)	1.60 (0.87–2.92)
Asian	2.02 (0.88–4.62)	2.52 (0.76–8.36)	3.69 (1.49–9.14)
Hypertension	1.74 (1.02–2.96)	1.84 (0.79–4.31)	3.04 (1.23–7.49)
Diabetes mellitus	2.13 (1.32–3.42)	1.37 (0.67–2.81)	1.52 (0.80–2.86)
LDL cholesterol (mmol/L)*	1.29 (1.07–1.57)	1.25 (0.94–1.67)	1.08 (0.83–1.41)
ESR (per 10 mm/h)*	1.20 (1.05–1.37)	1.09 (0.88–1.35)	1.23 (1.04–1.46)

CI indicates confidence interval; ESR, erythrocyte sedimentation rate; ICAS, intracranial arterial stenosis; LDL, low-density lipoprotein; OR=odds ratio.

*Ratio per unit increase.

studied populations, definition of ICAS, and used imaging modalities. Thus far, large studies on the prevalence, distribution, and risk factors predisposing ICAS have been mainly performed in Asian stroke populations.[20–22] Relatively high prevalence of 26% to 54% was observed in studies of different Asian populations of stroke patients. In contrast, limited studies have evaluated the prevalence of ICAS in Europe.[4,5,6] In a multicenter European study by Weimar et al,[6] using various imaging modalities, symptomatic ICAS of ≥50% was observed in 6.5% of the evaluated stroke patients. In contrast, the results of the present study reveal a lower prevalence of symptomatic ICAS ≥50% in European stroke patients (3%). The difference in prevalence can be partly explained by the inclusion of a higher proportion of patients with TIA and the exclusion of patients with severe ischemic stroke (Rankin score <4 at discharge) in the current study.

Observed prevalence and distribution of ICAS are also influenced by the applied imaging modality.[23] In general, transcranial Doppler is more operator-dependent and obtained results vary according to operator skills. Moreover, transcranial Doppler is suggested to be less sensitive than CTA for the detection of ICAS in the posterior circulation.[23,24] However, in Asian patients with TIA and ischemic stroke, the anterior circulation seems to be the predilection site in the distribution of ICAS irrespective of the imaging modality.[25–27] Distribution of ICAS reported in European stroke patients has been less consistent. Using either digital subtraction angiography or ultrasonography for primary detection, Mazighi et al[4] reported a similar distribution of ICAS in the anterior and posterior circulations. In contrast, using Doppler/duplex ultrasonography in 99% of the studied patients, Weimar et al[6] observed a higher prevalence of ICAS in the anterior (77%) versus the posterior (23%) circulation. Using MDCTA in current study, the majority of ICAS (67%) was located in the posterior circulation. The lower proportion of ICAS in the posterior circulation in the previous European studies may be attributable to a detection bias, because investigators have mainly relied on ultrasonography. Of note, detection of ICAS in the posterior circulation may be an important prognostic determinant because these lesions have been associated with a high risk of recurrent stroke.[1,28,29]

Composition of ICAS Lesions

Thus far, limited imaging studies have evaluated the composition of ICAS lesions. CT brain studies have reported a predominant presence of calcification in the proximal arteries but did not combine the evaluation of ICAS and plaque calcification with MDCTA.[30,31] Our findings confirm the presence of calcified ICAS lesions in the proximal intracranial arteries. However, a majority of noncalcified ICAS lesions was demonstrated in the distal arteries, which would be neglected on CT of the brain. This implicates that absence of calcification on CT of the brain does not exclude the presence of ICAS in the distal arteries. In line with the results of the current study, a previous postmortem histological analysis of atherosclerotic plaque composition in the medial cerebral artery has demonstrated calcification in only a minority of the specimens (31 of 111; 28%).[32]

The low prevalence of calcified ICAS lesions on MDCTA in the distal intracranial arteries suggests a different pathophysiology of atherosclerotic disease in the proximal and distal intracranial arteries. The intracranial arteries show significantly greater antioxidant enzyme activities than the extracranial arteries.[8] The greater activity of antioxidant enzymes in intracranial arteries may contribute to a greater resistance to atherogenesis. This antiatherogenic activity decreases significantly in older age, coinciding with accelerated atherogenesis.[8] Consequently, with age, intracranial arteries may respond with accelerated atherogenesis as their antioxidant protection decreases more significantly than that of the extracranial arteries. In the current study, the higher prevalence of extracranial stenosis in patients with ICAS supports the loss of protective antioxidant capacity in the extracranial arteries at a younger age. In line with this observation, higher plasma C-reactive protein levels have been previously noted in patients with extracranial stenosis as compared to those with isolated medial cerebral artery stenosis.[33]

Furthermore, with age, plasma LDL becomes more susceptible to oxidation.[34] The oxidative modification of LDL therefore may play a key role in this atherogenic process through inflammatory reactions.[35,36] The presence of mainly noncalcified ICAS lesions in the basilar artery and arteries beyond the circle of Willis in the current study might be a reflection of this accelerated atherogenesis.

Risk Factors Associated With ICAS

A number of traditional risk factors for atherosclerotic disease have been previously related to ICAS.[3,9,37] In addition, high-sensitivity C-reactive protein, a marker of inflammation, is associated with recurrent ischemic events in the territory of the stenotic artery in stroke patients with ICAS.[5] Also, ESR was shown to be independently associated of with the presence of ICAS in a South Asian stroke population.[10] In the present study of European patients, ICAS lesions were associated not only with the traditional risk factors including hypertension, diabetes, and LDL cholesterol but also with ESR. Importantly, the association of ESR with ICAS remained significant even after adjustment for the time since onset of symptoms. Thereby, the contribution of the acute phase reaction as a cause of ESR elevation was made less probable. As a result, an independent association was identified between ESR and LDL cholesterol with the presence of ICAS and, more importantly, with the presence of noncalcified ICAS lesions in patients with a recent TIA or ischemic stroke. These findings may indicate a prominent role for inflammation in intracranial atherogenesis.[35,36]

Study Limitations

The design of the present study is cross-sectional. The prognostic values of the presence, distribution, and calcification of ICAS lesions in patients with ischemic stroke or TIA remain to be determined in follow-up studies. The pathophysiological mechanisms initiating intracranial atherosclerosis were not evaluated. However, the predisposing risk factors and degree of calcification of ICAS support the current

hypothesis on the delayed development of intracranial atherosclerosis.

In the current study, the association of ICAS with the ESR was investigated as a marker of inflammatory processes in the atherosclerosis of the intracranial arteries. However, the ESR is only an indirect indicator of inflammatory processes and could be increased because of comorbidity. We did not exclude patients with comorbidity associated with ESR elevation to avoid additional bias. Evaluation of additional inflammatory markers such as high-sensitivity C-reactive protein and interleukins could have provided additional data on the role of inflammatory processes in intracranial atherosclerosis. Finally, the ESR was only measured at a single time point and during the acute phase in some of the patients.

Conclusions

It has been suggested that atherosclerosis in the extracranial carotid artery is the primary source of ischemic stroke in white patients.[38] We observed a low prevalence of ICAS in the current study population of predominantly white ethnicity. However, most ICAS were observed in the posterior circulation, a location associated with a high risk of recurrent stroke.[1,28,29]

Mainly noncalcified ICAS lesions were observed in distal intracranial arteries. A strong association of LDL cholesterol and ESR was identified with the presence of ICAS and, more importantly, with the presence of noncalcified ICAS lesions in patients with a recent TIA or ischemic stroke. Accordingly, in intracranial atherogenesis, a prominent role is indicated for inflammation. Further research on noninvasive analysis of plaque components in ICAS lesions could improve understanding of the pathophysiology of intracranial atherosclerosis. The additional evaluation of intraplaque hemorrhage using high-resolution MRI, which is likely to convey strong prognostic value for recurrent stroke, may be of particular interest.[39]

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Disclosure

None.

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