

Aetiologies, clinical and vascular findings in symptomatic and asymptomatic carotid artery occlusion

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Summary

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Carotid artery occlusion (CAO) is an important finding in stroke with different aetiologies. It most often occurs at the carotid bifurcation, less often at the intracranial portion of the internal carotid artery and rarely at the common carotid artery. Clinical presentations range from asymptomatic cases to severe neurological deficits or death. Cerebrovascular events in carotid artery occlusion have been attributed to either thromboembolic and/or haemodynamic mechanisms. The sensitivity and specificity of newer Duplex ultrasound techniques were found to be high for the detection of carotid artery occlusion in case studies as well as in a meta-analytic review.

In this retrospective study of 174 consecutive patients with either symptomatic (SCAO) or asymptomatic carotid artery occlusion (ACAO) detected by neurosonological methods, the clinical characteristics and findings of diagnostic tests are summarised. The vast majority of patients in the ACAO group was examined because of a cerebrovascular event *not* related to the occluded carotid artery. Clinical data included sex, age, vascular risk factors, medication, site of occlusion, stroke syndrome according to the Oxford Community Stroke Project, result of digital subtraction angiography (DSA) in selected cases, presumed aetiology after diagnostic work-up and outcome. Carotid artery occlusion was symptomatic in 67% and asymptomatic in 31%. The high proportion of asymptomatic carotid artery occlusions contributed to a relatively good outcome of carotid artery

occlusion, leaving 69% with no or only slight disability (Rankin scale 0–2). In patients with proximal carotid artery occlusion who had undergone digital subtraction angiography the rate of near occlusions was 14%. Atherosclerosis was the most common cause of carotid artery occlusion (78%), followed by dissection (5%) and emboli from infracarotid structures (2%). In 14% no aetiology could be identified after complete diagnostic work-up. Comparisons made between the symptomatic and asymptomatic group mainly revealed different aetiologies. Patients with asymptomatic carotid artery occlusion usually had atherosclerosis, while in symptomatic carotid artery occlusion further aetiologies like dissection and embolism were observed. Symptomatic carotid artery occlusion did not differ from asymptomatic carotid artery occlusion regarding collateral pathways, contralateral high-grade stenosis, vascular risk factors or demographic data. The result that patients with asymptomatic carotid artery occlusion were more often on treatment with oral anticoagulants could *not* be explained by an overrepresentation of patients with atrial fibrillation. This unexpected finding needs to be confirmed by further studies. The relatively high rate of near occlusions missed by neurovascular ultrasound may be due to the condition that angiography was only performed in doubtful cases. Nevertheless, the fact that the rate was still over 5% if patients without angiography were included raises the question whether the rate of false-positive carotid artery occlusion ultrasound findings might be higher than previously reported.

Keywords: carotid artery occlusion; asymptomatic; stroke; duplex; ultrasound

Introduction

Carotid artery occlusion (CAO) is an important mechanism of stroke with different aetiologies. Clinical presentations range from asymptomatic cases to severe neurological deficits or death [1–3]. Carotid artery occlusion most often occurs at

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the proximal, i.e. cervical or distal (intracranial) portion of the internal carotid artery (ICA). Thromboembolic and haemodynamic mechanisms have been invoked in the pathogenesis of cerebral ischaemic events related to carotid occlusion [1, 4]. The sensitivity and specificity of Duplex ultrasound, especially colour-coded Duplex sonography, was found to be high for the detection of carotid artery stenosis and carotid artery occlusion [5–7] in case studies as well as in a meta-analytic review [8]. Nevertheless, the main limitation of Doppler and Duplex ultrasonography is its accuracy in differentiating occlusion from near-occlusion. False-positive rates of around 5% compared to digital subtraction angiography (DSA) as gold standard have been reported by Kirsch et al. [5] and 0% at the expense of a decrease in specificity (from 100 to 78%) with the use of slow-flow sensitivity [7]. Ultrasound diagnosis of carotid artery occlusion seems to be less exact in clearly symptomatic patients compared with asymptomatic or remotely symptomatic patients [9]. One goal of this study was to ascertain whether carotid artery occlusion occurs without causing symptoms and to look for the most frequent aetiologies of carotid artery occlusion apart from atherosclerosis. In addition we addressed the question of whether SCAO patients differ in aetiology or neurosonological characteristics from ACAO patients.

Subjects and methods

Among 4500 patients seen in our ultrasound laboratory within a three-year period we identified 174 (4%) with carotid artery occlusion. We routinely examine in- and outpatients with acute and chronic cerebrovascular symptoms, investigate patients with carotid bruits, dizziness or syncope of presumed vascular aetiology, follow up patients after carotid thrombendarterectomy and occasionally screen patients before heart operations. Clinical and vascular data were obtained from ultrasound reports and clinical charts. Patients' demographic data included sex, age and vascular risk factors. Clinical data included site of occlusion, presence or absence of symptoms attributable to carotid artery occlusion, stroke syndrome according to the Oxford Community Stroke Project (OCSF) [10], medication, need for catheter-angiography, presumed aetiology after diagnostic work-up and clinical outcome as measured by the modified Rankin scale.

Patients were considered to have asymptomatic carotid artery occlusion if there were no reports of symptoms or signs attributable to vascular

events in the territory of the occluded carotid artery. Symptomatic carotid artery occlusion was defined as occurrence of at least one transient ischaemic attack (TIA) or stroke attributable to carotid circulation on the same side prompting ultrasonography that showed no patency of the corresponding carotid artery. When carotid artery occlusion was bilateral, symptoms or signs on one side were sufficient for diagnosis of symptomatic carotid artery occlusion. Reliable information concerning presence or absence of symptoms and signs attributable to carotid artery occlusion was available in 156 of the patients (90%). Clinical presentations of symptomatic carotid artery occlusion were divided into retinal versus hemispheric, and TIA or amaurosis fugax (AF) versus infarction. In patients with hemispheric stroke, the OCSF classification was used to assess the anterior circulation syndromes such as TACS (total anterior circulation syndrome), PACS (partial anterior circulation syndrome) or LACS (lacunar syndrome). The cause of carotid artery occlusion was defined after complete diagnostic work-up (ultrasound findings, echocardiography, 24-h ECG, conventional angiography, MRA), which was selected by the treating neurologist. Carotid artery occlusion was attributed to dissection if findings on digital subtraction angiography and/or MR angiography corroborated typical ultrasound findings (tapering occlusion above the carotid bifurcation without signs of atherosclerosis). Embolic carotid artery occlusion was defined by hypoechogenic occlusion of the lumen without significant atherosclerotic changes and presence of a prothrombotic state or a cardiac source of embolism. The cause of carotid artery occlusion was considered to be unknown if two or more stroke aetiologies were identified or results were all negative.

Atherosclerosis-defining ultrasound characteristics were either significant thickening of the intima-media complex, presence of plaques, contralateral ICA-stenosis and collateral pathways or a combination of these. Most patients had been examined with Doppler ultrasonography (*EME Pioneer TC 2020*) and colour-coded Duplex ultrasonography (*Acuson 128 XP/10c*), the former being used for extra- and transcranial insonation, the latter for extracranial examination. Ultrasound criteria for carotid artery occlusion included absence of flow in the internal carotid artery above the carotid bifurcation combined with dampening of the common carotid artery (CCA) signal and features of collateral flow [11]. We did not apply ultrasound contrast agent to establish carotid artery occlusion. Occlusion of the intracranial

Table 1 Baseline characteristics of CAO patients.

age (years)	66.1 (SD ± 10.5)
male	69% (120/174)
female	31% (54/174)
vascular risk factors (median)	2
hypertension	56% (89/158)
smoking	59% (92/156)
hyperlipidaemia	38% (54/143)
diabetes	20% (32/156)
atrial fibrillation	10% (14/141)
coronary heart disease	43% (57/131)
antiplatelet therapy	60% (75/125)
catheter angiography	54% (76/141)
right carotid artery occlusion	47% (82/173)
left carotid artery occlusion	46% (80/173)
bilateral carotid artery occlusion	6% (11/173)
proximal internal carotid artery occlusion	89% (154/173)
distal internal carotid artery occlusion	7% (13/173)
proximal + distal internal carotid artery occlusion	1% (1/173)
common carotid artery occlusion	3% (5/173)

Table 2 Clinical presentation of carotid artery occlusion.

symptomatic	67% (107/156)
asymptomatic	31% (48/156)
unclear	1% (1/156)
infarction (hemispheric/retinal)	78% (83/105)
transient ischaemic attack / amaurosis fugax	22% (23/105)
partial anterior circulation syndrome	66% (46/70)
total anterior circulation syndrome	22% (23/70)
lacunar syndrome	7% (5/70)
not classifiable	3% (2/70)

internal carotid artery was diagnosed when the proximal internal carotid artery was patent but a high resistance profile and signs of collateralisation were present. In 10 patients (5.8%), where only Doppler sonography data were available, diagnosis of occlusion was made by absence of a Doppler signal and features of collateral flow. In patients who had undergone digital subtraction angiography near occlusion was rated as false-positive ultrasound finding. Disability after stroke was measured by the modified Rankin scale as given in the last available medical record. Statistical analyses were done either by Chi-square or Fisher's exact test. The baseline characteristics of the patients with carotid artery occlusion are shown in table 1.

Results

The clinical presentations of carotid artery occlusion are listed in table 2. Carotid artery occlusion was symptomatic in 67% and asymptomatic in 31% of 156 patients. The vast majority of patients in the ACAO group was examined because of acute cerebrovascular symptoms *not* related to the occluded carotid artery. In one patient (1%) the relation remained uncertain. In 78% of 105 symptomatic patients with involvement of the central nervous system (2 patients with isolated cranial nerve symptoms are excluded), clinical manifestations of carotid artery occlusion were a cerebral or retinal infarction and in 22% transient ischaemic attack or amaurosis fugax. The relative frequency of stroke syndromes was determined in patients with hemispheric infarction (n = 70). 66% had a PACS, 22% had a TACS, 7% had a LACS and 2 (3%) were not classifiable.

The aetiology and outcome of carotid artery occlusion are listed in table 3. Cerebrovascular atherosclerosis, defined by the above-mentioned ultrasound criteria, was the most common cause of carotid artery occlusion, being observed in 78% of 165 patients with available information. Less frequent aetiologies included ICA-dissection (5%), embolism from infracarotid thrombi (2%), moya moya (1%) or granulomatous disorder (1%). The source of embolic carotid artery occlusion in 2 of the 4 patients was cardiac. The other 2 had a prothrombotic state with morbus embolicus, in one of them caused by combined deficiency of protein S, protein C and antithrombin III.

No disability after carotid artery occlusion was found in 49% of the 165 patients for whom information was available. A slight disability (Rankin 1+2) was found in 20%, a moderate disability (Rankin 3+4) in 23%. Severe disability with full dependence or death occurred in 8% after carotid artery occlusion.

Comparing SCAO and ACAO patients (see table 4), atherosclerosis was significantly more common in asymptomatic carotid artery occlusion (98 vs 85%, p = 0.034). There was an association between asymptomatic carotid artery occlusion and oral anticoagulation: 34% on the ACAO group were taking an oral anticoagulant drug (usually phenprocoumon) at the time of diagnosis compared with 8% in the SCAO group (p = 0.001). No significant differences between the two groups were found in relation to a medication with aspirin, age, sex, side of occlusion, vascular risk factors, comorbidity, presence of collateral pathways (via ophthalmic and/or anterior communicating artery) or a contralateral high-grade stenosis. The rate of

Table 3 Aetiologies and outcome of carotid artery occlusion.

atherosclerosis	78% (128/165)
dissection	5% (8/165)
embolism	2% (4/165)
moya moya	1% (1/165)
granulomatous	1% (1/165)
undetermined	14% (23/165)
Rankin 0	49% (75/154)
Rankin 1 + 2	20% (31/154)
Rankin 3 + 4	23% (36/154)
Rankin 5 or death	8% (12/154)

Table 4 Comparison of aetiologies and other clinical factors.

	ACAO	SCAO	p
atherosclerosis	98%	85%	0.034*
dissection	0%	9%	0.051*
embolus	0%	5%	n.s.*
atrial fibrillation	44%	42%	n.s.**
oral anticoagulation	34%	8%	<0.001**
aspirin	32%	47%	n.s.**
history of TIA/AF	18%	43%	0.043**
contralateral high-grade stenosis	21%	18%	n.s.**
no collateral pathway	7%	13%	n.s.*

*Fisher's exact test; **Chi-square test.

near occlusion as found by catheter angiography was 14% in patients with proximal carotid artery occlusion.

Discussion

During the last years, considerable attention has been paid to diagnosis and treatment of internal carotid artery stenosis, while carotid artery occlusion has not received the same degree of interest, probably because it is mostly regarded as the *end-point* of an occlusive atherosclerotic disease. Most studies about carotid artery occlusion do not differentiate between symptomatic and asymptomatic occlusions and do not take into consideration the various aetiologies of carotid artery occlusion.

The high proportion of asymptomatic carotid artery occlusions (31%) in this selected population contributed to a relatively good outcome, leaving 69% with no or only slight disability (Rankin scale 0–2) after carotid artery occlusion. A low long-term stroke risk (0–4%), but a relevant annual cardiovascular mortality (6–9%) was seen in pa-

tients with unilateral carotid artery occlusion [12] and asymptomatic carotid artery occlusion [13].

As expected, the most frequent aetiology of carotid artery occlusion in this case series was atherosclerosis (78%). The next frequent aetiology was carotid dissection, a finding not reported in other series. Rare aetiologies were emboli and moya moya disease (one patient with asymptomatic carotid artery occlusion). Our results do not support the opinion [14] that cardiac emboli are an important cause of carotid artery occlusion, nor have we observed other recognised aetiologies, such as arteritis, trauma or radiation [15–17].

The unexpected result that patients with asymptomatic carotid artery occlusion were more often on treatment with oral anticoagulants at time of diagnosis could not be explained by an overrepresentation of patients with atrial fibrillation. This correlation needs to be confirmed by further studies. The high rate of near occlusions (14%) in patients with ultrasound finding of carotid artery occlusion as confirmed by digital subtraction angiography may be explained by the fact that angiography was only performed in doubtful cases.

We did not find other factors associated with asymptomatic carotid artery occlusion, such as age, sex, vascular risk factors, collateralisation or presence of a high-grade stenosis on the contralateral side. The main limitations of this study lie in the retrospective design, the limited investigation of collateral pathways and the lack of results from cerebral imaging methods which could have revealed silent infarctions.

Conclusion

Symptomatic carotid artery occlusion did not differ from asymptomatic carotid artery occlusion regarding collateral pathways, contralateral high-grade stenosis, vascular risk factors or demographic data. Patients with asymptomatic carotid artery occlusion usually have atherosclerosis, while in symptomatic carotid artery occlusion further aetiologies like dissection and embolism are observed. The rate of near occlusions missed by neurovascular ultrasound in clinical routine may be higher than previously thought.

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