The Prognosis of Carotid Siphon Stenosis

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SUMMARY We retrospectively reviewed the clinical course and angiograms of 15 patients with carotid siphon stenosis of 50% or greater. Fourteen had less than 50% stenosis at the origin of the ipsilateral extracranial internal carotid artery, and one had a greater degree of stenosis but underwent endarterectomy after an initial angiogram. Angiograms were examined for evidence of hemodynamic abnormalities in addition to residual lumen diameter. Seven patients initially had TIA, 5 had strokes, and 3 were asymptomatic. In an average follow-up of 51 months (range 4–123 months) subsequent cerebral ischemic events occurred in 6 (40%), but only 1 had a stroke with a persisting neurological deficit that could be directly attributed to the siphon stenosis. Stenoses were hemodynamically significant by angiography in 5 of 7 TIA patients, and only 1 of 5 stroke patients. The incidence of subsequent ischemic events in this study was similar to 2 previous studies of siphon stenosis, however in this study most of the events ipsilateral to the siphon stenosis were TIA or minor strokes. The association of hemodynamic angiographic abnormalities and initial TIA but not strokes suggests that the mechanism producing ischemic symptoms may differ in patients with TIA and stroke who have carotid siphon stenosis.

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CALCIFICATION of the carotid siphon or intracranial internal carotid artery occurs frequently, but significant stenosis of this segment is less common.1 Stenosis of the carotid siphon has been estimated to be about one-sixth as common as disease of the carotid bifurcation.2 Until recently, little was known about the natural history of stenosis of the carotid siphon. Two previous studies of siphon stenosis found that 30–40% of such patients had subsequent ischemic events, and more than half of these events were ipsilateral to the siphon stenosis.3,4 Clinical details of events occurring during follow-up were limited in both studies, and the angiographic data included only the site and severity of stenosis. Angiograms were not examined for collateral flow or hemodynamic significance of the siphon stenosis.

We therefore retrospectively reviewed clinical and angiographic information on 15 patients with carotid siphon stenosis of 50% or greater. Fourteen had less than 50% stenosis of the ipsilateral extracranial carotid and 1 had severe ipsilateral extracranial stenosis but underwent endarterectomy shortly after angiography. All angiograms were reviewed for hemodynamic as well as anatomic information and the results were correlated with the nature of initial and subsequent ischemic events.

Methods

Patients were identified by reviewing the angiography records of the neuroradiology department for the past 5 years for the diagnosis of stenosis of the carotid siphon. Those with greater than 50% stenosis of the ipsilateral extracranial internal carotid artery, ipsilateral carotid occlusion, or middle cerebral artery stenosis were excluded unless carotid endarterectomy was performed after angiography. The medical records were then reviewed for details of the initial clinical syndrome. Follow-up information was obtained directly from patients, hospital records, physicians, and family members. Angiograms were reviewed by the neuroradiologist (KRD) without knowledge of the clinical information or subsequent course. The site and residual lumen diameter of the stenotic lesion was recorded in each case. Stenosis between the ophthalmic artery and the terminal carotid bifurcation was considered supracholinoid. A lesion between the petrous portion of the carotid and the ophthalmic artery was designated cavernous. Lumen diameter was expressed as percent of the diameter of the nearest normal appearing segment below the site of stenosis. Residual diameter of the lumen was measured at the point of maximal narrowing on either the AP or lateral view.

Evidence of a hemodynamically significant stenosis included delayed filling of the middle cerebral artery (MCA) branches relative to the external carotid branches, prolonged crossflow through the anterior circle of Willis into the ipsilateral MCA and shift of the anterior cerebral watershed toward the middle cerebral artery territory after injection of the contralateral carotid.

Results

Table 1 summarizes angiographic and clinical information on each patient. Forty percent of the patients had either stroke or TIA during the follow-up period. Five patients died; 3 from presumed myocardial infarction, one from basilar thrombosis, and one from a cerebellar hemorrhage. Average follow-up was 51 months (range 4–123 mo). All but 3 patients (T3,S4,A3) were treated with either Coumadin, antiplatelet drugs or both. Three groups of patients can be identified based on initial clinical syndromes. Seven patients had TIA, 5 had strokes and 3 were asymptomatic.

TIA Group

Six of 7 patients with an initial TIA had at least 2 identical spells. Three patients (T1,T4,T7) had at least
2 types of spells, and none of the TIAs lasted more than an hour. In 3 patients the stereotypical spells were transient monocular blindness (TMB) and in 3, spells were transient hemispheric attacks (THA) involving speech difficulty, weakness, or numbness and weakness of the hand or arm. One patient with several episodes of TMB also had a single hemispheric attack (T4). In 2 cases there were other possible lesions that may have produced symptoms. One patient (T4) had stenosis of the extracranial internal carotid artery and underwent endarterectomy shortly after angiography. The other patient (T5) had a left parietal meningioma in addition to left siphon stenosis.

Five of the 7 patients with TIAs had evidence of impaired flow on angiography (T1,T2,T4,T6,T7). Two of 3 patients with multiple stereotyped hemispheric attacks (T1,T7) and all 3 patients with TMB (T2,T4,T6) had hemodynamically significant stenosis. The patient (T3) with a single ischemic episode lasting less than 1 hour did not have a hemodynamically significant stenosis.

Subsequent ischemic events occurred in 3 patients. In the first (T2) a single recurrent episode of TMB occurred 9 months later and then no further spells in 87 months. The second patient (T5) developed an ischemic deficit immediately after angiography that was identical to the initial TIAs. This cleared completely in 48 hours and he had no further symptoms. The third patient (T4) continued to have TIAs despite endarterectomy for an ipsilateral extracranial stenosis, and after 46 months developed a fixed deficit identical to his previous TIAs. When angiogramed at the time of the stroke he had the same hemodynamic changes noted on an angiogram done prior to endarterectomy, suggesting that the siphon stenosis was responsible for both initial and subsequent symptoms.

Two patients died during followup. 1 from a myocardial infarction (T7), and the other from a cerebellar hemorrhage 123 months after the initial evaluation (T4). The patients with recurrent symptoms (T2, T4,T5) were treated with antiplatelet drugs, and 1 also received Coumadin (T4). Four patients with no further symptoms were treated with Coumadin (T1,T7), antiplatelet drugs (T6), or neither (T3).

**Stroke Group**

All 5 strokes that led to the diagnosis of siphon stenosis were in the distribution of the left carotid artery. Onset of the stroke was sudden and maximal in 3 (S2, S4, S5) and may have stuttered initially in 2 (S1, S3). Only 1 (S4) was preceded by a TIA, and only a single TIA occurred in that patient. Deficits were mild or moderate and improved or resolved over days to weeks. Unlike the TIA group, 4 of 5 patients with strokes did not have a hemodynamically significant siphon stenosis.

Two of the 5 (S1, S4) had a second stroke during the followup period and 1 died (S4). The second stroke occurred 2 years later on the same side as the siphon stenosis in one patient (S1). Repeat angiography showed a new severe stenosis (residual lumen diameter <1 mm) of the extracranial internal carotid artery. Fatal basilar thrombosis occurred in another patient (S4) 34 months after his initial left hemisphere stroke. In both cases the second stroke was probably unrelated to the siphon stenosis. None of the patients in the stroke group had TIAs during the followup period.

Three patients were treated with antiplatelet drugs (S1, S2, S5) including the patient with a second stroke and a new extracranial internal carotid stenosis (S1). The patient with a hemodynamically significant stenosis on angiography (S3) was treated with Coumadin and had no further ischemic symptoms. One patient in this group (S4) received neither Coumadin or antiplatelet drugs. Basilar thrombosis occurred in this patient.

**Asymptomatic Group**

Three patients had siphon stenosis in the absence of ischemic symptoms. Reasons for angiography included a self-audible bruit contralateral to the siphon stenosis, an asymptomatic bruit, and TIAs related to the contralateral carotid. There was angiographic evidence of hemodynamically significant siphon stenosis in one of the 3 cases (A2).

No strokes occurred during followup. One of the 2 patients with stenosis that was not hemodynamically significant later developed transient monocular blindness ipsilateral to the stenosis (A3). Angiography 9 months after the initial study showed no change in the stenosis, and poor filling of the ophthalmic artery. Two patients in this group (A1,A2) were treated with antiplatelet agents, and 2 (A1,A3) underwent endarterectomy on the side contralateral to the siphon stenosis.

**Angiographic Findings**

Seven of the 15 patients had a hemodynamically significant stenosis on angiography. TIAs were the initial manifestation in 5 (T1,T2,T4,T6,T7), stroke in 1 (S3), and 1 was asymptomatic (A2). Two of these 7 patients later had additional ischemic events in the same territory and of the same type (T2,T4). In 8 patients without hemodynamically significant siphon stenosis, 1 (T5) had a stroke in the same distribution after angiography, 1 (A3) had TMB, and 6 had no symptoms or had symptoms due to lesions other than the siphon stenosis. In 3 of the 7 patients with a hemodynamically significant siphon stenosis (T1,S3,A2), the contralateral carotid filled both anterior cerebral arteries (ACA) and crossfilled into the ipsilateral middle cerebral artery (MCA). None of these patients had subsequent ischemic symptoms. There was no crossfilling into the ipsilateral MCA in 4 angiograms (T2,T4,T6,T7). Two of these patients later had ischemic symptoms (T2,T4).

**Discussion**

In 15 patients with carotid siphon stenosis and less than 50% extracranial internal carotid artery stenosis followed for an average of 51 months, TIAs occurred in 3 (20%) and stroke in 4 (27%). Most patients with TIAs as the initial symptom had a hemodynamically
<table>
<thead>
<tr>
<th>Pt</th>
<th>Age</th>
<th>Site of Stenosis</th>
<th>Percent Stenosis</th>
<th>Hemodynamic Significance</th>
<th>Collateral</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>30</td>
<td>Left supraclinoid</td>
<td>57%</td>
<td>Yes</td>
<td>R carotid fills both ACAs and into L MCA. Watershed shift.</td>
<td>15 spells of numbness and weakness of R hand. Also aphasia, numbness R hand.</td>
</tr>
<tr>
<td>T2</td>
<td>59</td>
<td>Left cavernous</td>
<td>78%</td>
<td>Yes</td>
<td>R carotid fills both ACAs but no crossfilling. Watershed shift.</td>
<td>Several spells of iris-like visual loss on L.</td>
</tr>
<tr>
<td>T3</td>
<td>61</td>
<td>Right cavernous</td>
<td>63%</td>
<td>No</td>
<td>No crossfilling into R MCA. No watershed shift.</td>
<td>Dysarthria, numbness and weakness L F, A, L.</td>
</tr>
<tr>
<td>T4</td>
<td>63</td>
<td>Left cavernous</td>
<td>63%</td>
<td>Yes</td>
<td>No crossfilling into L MCA. No watershed shift.</td>
<td>Aphasia, weakness R arm, leg × 1 hr. Several spells L TMB.</td>
</tr>
<tr>
<td>T5</td>
<td>74</td>
<td>Left supraclinoid</td>
<td>62%</td>
<td>No</td>
<td>R carotid fills both ACAs, no crossfilling into L MCA. No watershed shift.</td>
<td>2 spells of aphasia, numbness, weakness R hand. L parietal mass.</td>
</tr>
<tr>
<td>T6</td>
<td>74</td>
<td>Left cavernous</td>
<td>75%</td>
<td>Yes</td>
<td>No crossfilling into L MCA. No watershed shift.</td>
<td>2 spells of blurred vision L eye. L periorbital pain.</td>
</tr>
<tr>
<td>T7</td>
<td>74</td>
<td>Left cavernous</td>
<td>80%</td>
<td>Yes</td>
<td>R carotid fills both ACAs, but no crossfilling into L MCA. Watershed shift.</td>
<td>Many spells weakness of R arm. Also brief spells of garbled speech.</td>
</tr>
<tr>
<td>S1</td>
<td>66</td>
<td>Left supraclinoid</td>
<td>67%</td>
<td>No</td>
<td>R carotid fills both ACAs, no crossfilling into L MCA. No watershed shift.</td>
<td>Stuttering onset of slurred speech, weakness F, A, L.</td>
</tr>
<tr>
<td>S2</td>
<td>78</td>
<td>Left cavernous</td>
<td>75%</td>
<td>No</td>
<td>No crossfilling into L MCA. No watershed shift.</td>
<td>Sudden onset of numbness, weakness R arm, aphasia.</td>
</tr>
<tr>
<td>S3</td>
<td>43</td>
<td>Left cavernous</td>
<td>83%</td>
<td>Yes</td>
<td>No crossfilling into L MCA. No watershed shift.</td>
<td>Onset over 4 hrs. of weakness, numbness R arm.</td>
</tr>
<tr>
<td>S4</td>
<td>62</td>
<td>Left supraclinoid</td>
<td>63%</td>
<td>No</td>
<td>R carotid fills both ACAs, no crossfilling into L MCA. No watershed shift.</td>
<td>Sudden onset slurred speech, weakness R F, A, L. One TIA 3d prior.</td>
</tr>
<tr>
<td>S5</td>
<td>66</td>
<td>Left cavernous</td>
<td>57%</td>
<td>No</td>
<td>No crossfilling into L MCA. No watershed shift. R ICA occluded.</td>
<td>Sudden onset of weakness R arm, leg, aphasia.</td>
</tr>
<tr>
<td>A1</td>
<td>64</td>
<td>Right cavernous</td>
<td>71%</td>
<td>No</td>
<td>No crossfilling into R MCA. No watershed shift. L ICA stenotic at origin.</td>
<td>Audible bruit.</td>
</tr>
<tr>
<td>A2</td>
<td>57</td>
<td>Right cavernous</td>
<td>71%</td>
<td>Yes</td>
<td>L carotid fills both ACAs and into R MCA. Watershed shift.</td>
<td>Asymptomatic bruit.</td>
</tr>
<tr>
<td>A3</td>
<td>59</td>
<td>Right cavernous</td>
<td>50%</td>
<td>No</td>
<td>No crossfilling into R MCA. No watershed shift. L ICA ulcerated plaque at origin and L MCA embolus.</td>
<td>None related to right carotid. One spell of paresthesias R face and foot.</td>
</tr>
</tbody>
</table>

L = left; R = right; ACA = anterior cerebral artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; ICA = internal carotid artery; F = face; a = arm; l = leg.

significant stenosis on angiography and those with stroke did not.

Two previous studies of siphon stenosis differed from ours because they included patients with significant tandem lesions.3,4 Despite this difference, the overall incidence of ischemic events during followup in both studies was similar to ours (table 2), and most of the ischemic events in all 3 studies occurred ipsilateral to the siphon stenosis. No clinical information regarding the nature or severity of strokes during followup was given in the study by Marzewski et al.,2 and Craig et al4 state only that 5 strokes ipsilateral to a siphon stenosis were fatal. The results in the latter study clearly differ from the more benign outcome in our patients. Five of our patients had subsequent ischemic events ipsilateral to the siphon stenosis, but only 1 disabling stroke occurred that was directly related to the siphon stenosis. TMB alone occurred in 2 patients, a minor stroke in 1, and a stroke associated with a severe extracranial stenosis in another. All but 2 of our patients were treated with either antiplatelet agents or anticoagulants, and this may have influenced the severity of subsequent ischemic events. Mortality in all 3 studies of siphon stenosis was strikingly high (table 2).
Most deaths were due to cardiac disease or presumed cardiac disease, similar to patients with other forms of cerebrovascular disease.5,6

Neither previous study of siphon stenosis attempted to correlate angiographic information with clinical syndromes and outcome. Craig et al4 included data on other sites of carotid stenosis or occlusion but did not assess hemodynamic significance or crossflow. The tortuous course of the intracranial internal carotid artery makes measurement of the residual lumen diameter at the site of maximal stenosis difficult, since it may not be well visualized by the usual AP or lateral views. Delayed flow in the internal carotid artery relative to the external carotid artery may provide additional evidence of severe stenosis and may be more reliable than direct measurements of the residual lumen. This might explain why the nature of initial ischemic events (TIA vs stroke) correlated poorly with the measured degree of stenosis, but correlated well with hemodynamic angiographic changes. Severity of stenosis ranged from 57% to 80% in both the TIA and stroke groups, and ranged from 50% to 71% in asymptomatic patients. In contrast, there was angiographic evidence of slow flow in 5 out of 7 patients with TIs but only 1 of 5 with stroke as the initial event. Too few strokes occurred during followup to correlate hemodynamic changes with outcome, but it is of interest that the only persistent stroke occurring during followup ipsilateral to a siphon stenosis was in a patient with hemodynamically significant stenosis and poor crossflow through the anterior communicating artery from the opposite carotid.

The clinical-angiographic correlation in these patients suggests that mechanisms underlying ischemic events may differ in the TIA and stroke groups. TIs were mostly multiple, stereotyped, and associated with angiographic evidence of a hemodynamically significant stenosis. Strokes were sudden in onset or developed over hours. They were preceded by TIs in only 1 patient, and most did not have hemodynamically significant stenoses. An embolic mechanism was suspected. One possible explanation for the different angiographic findings in the TIA and stroke groups in that siphon stenosis may be detected earlier when embolic stroke occurs from either the carotid siphon lesion or a more proximal source. When the lesion progresses to severe stenosis with slow flow on angiography...
ography, TIAs may be the most common initial manifestation. Pessin et al.7 divided strokes associated with stenosis or occlusion of the extracranial internal carotid artery into 2 groups based on angiographic criteria; an embolic or suspected embolic group, and a non-embolic group. In the non-embolic group, 73% had TIAs and 41% had angiographic evidence of a hemodynamically significant stenosis in the internal carotid artery. In the embolic or suspected embolic groups, strokes were more severe but few were heralded by TIAs and none had hemodynamically significant stenoses. Thus, 2 subgroups of patients may be identified with vascular lesions at the same site but different mechanisms producing ischemic symptoms. These subgroups may differ in prognosis or response to therapy.

In our study there was no definite correlation between clinical or angiographic data and subsequent ischemic events ipsilateral to the siphon stenosis, but the number of events during followup was small. We suggest that future studies of prognosis in patients with vascular disease include angiographic analysis of hemodynamic significance and patterns of collateral flow to assess whether hemodynamic abnormalities and crossflow are important factors predisposing to recurrent ischemic events.

References


Spontaneous History of Asymptomatic Internal Carotid Occlusion

M. HENNERICI, M.D., H.-B. HÜLSBÖMER, W. RAUTENBERG, M.D., AND H. HEFTER, PH.D.

SUMMARY Forty-nine patients with ICA occlusion, who presented without any neurological signs or symptoms, were prospectively followed for an average of 31.2 months. Eight patients (16%) suffered a stroke during follow-up, of which five were within the vascular territory of the occluded artery — 5 patients (10%) developed TIAs 4 of which were ipsilateral to the occluded artery. Non-invasive vascular follow-up did not reveal a progression of extracranial arterial disease in the majority of later symptomatic patients. Twenty-three patients (46.9%) died during follow-up, coexisting coronary artery disease being the major cause of death.

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IN A RECENT PAPER Cote et al1 reported a study of 47 patients with occlusion of the internal carotid artery (ICA), who presented with transient ischemic attacks (TIA) or mild strokes and were identified by angiography during the Canadian Cooperative Study.2 Since none of them were submitted to extra-intracranial (EC/IC) bypass surgery, their spontaneous risk of repetitive cerebral infarction could be studied prospectively. Among the literature reviewed by these authors, this was the first published series of a large group of patients without major neurological deficits studied prospectively. Their results indicated that the risk of subsequent cerebral infarction ipsilateral to an occluded carotid artery was comparable to the general risk of patients suffering from TIAs indicating that ICA occlusion was not a stable situation, as might have been suggested.

The present paper attempts to define whether patients with symptomless ICA occlusions have a better prognosis. Such patients have been selected in a large prospective series since 1977.2-4 The risk of stroke in this group of asymptomatic patients with obstructive extracranial arterial disease (EAD) has been shown to be rather small, although the probability of EAD progression was high as was the mortality rate. This was mainly due to frequent coexisting silent coronary artery disease (CAD). The present comparison of both prospective series, including rather similar preliminaries, should provide further insight into the natural history of obstructive EAD and hence optimize therapy.
The prognosis of carotid siphon stenosis.
L R Wechsler, J P Kistler, K R Davis and M J Kaminski

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