Letters to the Editor

Prognosis of Carotid Siphon Stenosis

To the Editor:

In their interesting paper, Wechsler et al. confirmed that siphon stenosis carries a poor prognosis for life, as one-third of their patients were dead at the end of a 51-month follow-up. In a previous study involving 15 patients with siphon stenosis of > 30% (without proximal atherosclerosis of equal or greater severity), we also found a high mortality. Our series now has 22 patients (12 men, 10 women; mean age 60 years) who have been followed for an average of 40.4 months. The mortality rate is high (1 stroke death, 6 cardiac deaths) and is similar to the previously reported rates, but ipsilateral stroke is higher (Table 1). One-third of the patients could not resume prior activity. There is a striking difference between patients with associated proximal internal carotid artery atherosclerosis and those group; over 80% of all delayed events occurred in the former group. These findings suggest that 1) siphon stenosis in the context of associated proximal atherosclerosis is a marker of severe atherosclerotic disease and bears a very poor prognosis, and 2) isolated siphon stenosis may correspond to another type of atherosclerotic disease, although risk factors do not seem to differ. It is not known at the present time how often isolated siphon stenosis merits the term “atherosclerotic” in the absence of pathological studies.

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Table 1. Mortality and Delayed Ipsilateral Stroke in Siphon Stenosis

<table>
<thead>
<tr>
<th>Author (reference)</th>
<th>Mortality</th>
<th>Ipsilateral stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marzewski et al (3)</td>
<td>12.8%/year</td>
<td>2.3%/year</td>
</tr>
<tr>
<td>Craig et al (4)</td>
<td>17.2%/year</td>
<td>7.6%/year</td>
</tr>
<tr>
<td>Wechsler et al (1)</td>
<td>7.8%/year</td>
<td>4.7%/year</td>
</tr>
<tr>
<td>Personal series</td>
<td>9.5%/year</td>
<td>8.1%/year</td>
</tr>
</tbody>
</table>

References


The following letter is in reply:

To the Editor:

The report by Dr. Bogousslavsky of outcome in 22 patients with carotid siphon stenosis extends his previous data and adds to the published information on the prognosis of this lesion. The ipsilateral stroke rate was similar to that in our study, and as in our study the majority of ipsilateral strokes were not disabling. In contrast, 5 of 11 strokes ipsilateral to the siphon stenosis occurring during follow-up in the study by Craig et al were fatal. Although 10% of their patients had “tandem” lesions, none of the strokes occurred in this group. The reasons for this discrepancy are not immediately clear. Mortality has been uniformly high in all studies of siphon stenosis, mostly from cardiac disease.

We agree with Dr. Bogousslavsky that the pathology of “isolated” siphon stenosis may differ from atherosclerosis at the carotid bifurcation. Pathological studies observe differences in the histological features of atherosclerosis at these two locations. Although siphon stenosis is occasionally “isolated” when initially discovered, extracranial stenosis may appear later and contribute to subsequent ischemic events. This occurred in one of our patients who developed transient ischemic attacks and ipsilateral stroke associated with a new severe extracranial stenosis 53 months after an initial angiogram. Dr. Bogousslavsky’s results suggest that prognosis and possibly pathology differ depending upon whether siphon stenosis is associated with extracranial stenosis. This distinction is somewhat artificial. Although other causes of siphon stenosis no doubt occur, it may be a component of generalized atherosclerosis despite little or no extracranial stenosis when the siphon lesion is initially discovered.

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Aspirin and the Kidneys in Patients With Cerebral Ischemia

To the Editor:

The acceptance of aspirin therapy for prevention of cerebral ischemia is based on positive results of several large clinical trials. The usual dose was 1000–1500 mg/day. It is now known that this dose of aspirin is far greater than that needed to inhibit platelet aggregation in normal subjects, where doses of 20–50 mg daily inhibit platelet aggregation and TXA2 synthesis, but for the moment there is no convincing evidence from comparative studies that any dose of aspirin is more or less effective than another.

Several recent reports emphasize the adverse effects of aspirin, indomethacin, and other nonsteroidal anti-inflammatory agents (NSAIAs) on renal function (sodium retention, impairment of water excretion,
Prognosis of carotid siphon stenosis.
J Bogousslavsky

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