Headache and Transient Ischemic Attacks

BY ALAN B. GRINDAL, M.D.,* AND JAMES F. TOOLE, M.D.

Abstract: Headache occurring in association with episodes of transient cerebral ischemia has not been emphasized as part of the symptom complex.

In an effort to ascertain the frequency and characteristics of these headaches and to determine whether there is any localizing, diagnostic, or prognostic significance to them, the records of 240 TIA patients were reviewed.

We concluded that headache is a prominent symptom in 25% of patients with TIA and that there were no distinguishing population characteristics, arteriographical findings or prognostic features among patients whose TIAs were accompanied by headache. The locations and temporal relationships of headache occurring in cases of carotid and vertebrobasilar TIA also were compared.

Additional Key Words: amaurosis fugax, carotid ischemia, vertebrobasilar insufficiency, subclavian steal

Headache is a frequent premonitory symptom of cerebral ischemia.1,4 Andrell1 reported headache as an early symptom in three of nine cases of internal carotid thrombosis. This association was later confirmed by Fisher2 and Wells.3 Similar observations have been made with regard to vertebrobasilar insufficiency.5 Despite these reports, the occurrence of headache in association with other symptoms of transient cerebral ischemia has received little attention.

The objectives of this communication are: (1) to study the frequency, location, and temporal relationships of headaches which occurred in association with other symptoms of transient cerebral ischemia, and (2) to compare such patients in terms of population characteristics, angiographical findings, and prognosis with the larger TIA population from which these cases were drawn.

Methods

The records of 240 Caucasian patients with TIA admitted to this hospital between 1964 and 1973 were reviewed. All patients had at least one TIA within one month of hospitalization, and all received aortic arch and four-vessel selective arteriography. Those cases with a history of headache, cranial, facial, or neck pain temporarily related to other symptoms of transient cerebral ischemia were selected for study. Patients with light-headedness or cranial or facial numbness, as well as those in whom the relationship between headache and TIA was unclear, were not included. Also noted were those cases in which a definite negative history of headache was obtained.

From the entire population of 240 cases, 58 patients were selected. Based on clinical and angiographical findings, 56 patients were divided into two groups depending upon whether their symptoms were felt to arise from the carotid or vertebrobasilar circulation. In the remaining two cases the distribution of the TIA could not be determined.

One hundred and sixty of these 240 patients were the subject of a previous study in which age, sex, incidence of hypertension, distribution of angiographical lesions and prognosis were determined.5 This population of 160 was compared with the headache patients within this group (40) to determine if any significant difference in these parameters existed among patients whose TIAs are associated with headache.

Results

There appears to be no significant difference in age, sex, incidence of hypertension, distribution of arteriographical lesions, or prognosis in patients whose TIAs are manifested by headache (table 1).
In approximately 25% of patients with TIA headache is a prominent symptom and is the presenting complaint in nearly one-third. Because of the retrospective nature of this study and the fact that in only 50 (21%) patients was a definite negative history of headache obtained, this report will probably underestimate the true frequency of this association. We feel, however, that if headache were a principal symptom of the ischemic episode, it would have been included in the recorded history.

Among patients with TIAs in the carotid distribution, there was no clear relationship between angiographical data and the location, intensity, or duration of the headache. Headache was most commonly reported as nonlocalized or generalized in location (13 patients). However, in 19 of the remaining 20 cases, the headache was predominantly frontal or retro-orbital, being ipsilateral to the symptomatic carotid in nine, contralateral in two, and bifrontal in six (table 2). Of the five patients with amaurosis fugax and headache, four had ipsilateral frontal or orbital pain following the disturbance of vision.

Headache was rarely the initial symptom of carotid transient ischemia and commonly had its onset either in association with other neurological symptoms or following the resolution of the attack. Headache was a constant feature in only one-third of those patients who experienced more than one carotid TIA.

The headache occurring among patients with transient vertebrobasilar symptoms was primarily occipital or nuchal in location (15 of 23) (table 3). The headache was nonlocalized in five and bifrontal in two. As with carotid TIAs, headache tended to occur in association with or after the onset of accompanying neurological symptoms. In contrast to the carotid group, however, headache was a constant feature in the majority of patients with recurrent vertebrobasilar insufficiency. Six patients with subclavian steal syn

| Number | 160 | 40 |
| Age (mean) | 57 | 56.4 |
| Length of follow-up | 3.5 years | 3.5 years |
| Sex | M-107 F-53 | M-26 F-14 |
| Hypertension | 102/160 - 64% | 21/40 - 52.5% |
| Site of lesions | | |
| Carotid | 112 - 70.0% | 25 - 62.5% |
| One carotid | 40 - 25.0% | 9 - 22.5% |
| Bilateral carotid | 43 - 26.9% | 9 - 22.5% |
| Carotid-vertebrobasilar | 26 - 16.3% | 7 - 17.5% |
| Carotid-subclavian | 3 - 1.9% | 1 - 2.5% |
| Vertebrobasilar | 13 - 8.1% | 5 - 12.5% |
| Subclavian | 16 - 10.0% | 5 - 12.5% |
| Intracranial | 13 - 8.1% | 3 - 7.5% |
| Normal | 6 - 3.8% | 1 - 2.5% |
| Outcome | | |
| Asymptomatic | 90 - 56.3% | 25 - 62.5% |
| Continued TIAs | 22 - 13.8% | 5 - 12.5% |
| Cerebral infarct | 31 - 19.4% | 6 - 15.0% |
| Death | 23 - 14.4% | 4 - 10.0% |

### Table 2

**Headache With Carotid TIA**

<table>
<thead>
<tr>
<th>Location of headache</th>
<th>Total</th>
<th>Age (mean)</th>
<th>Sex: Male</th>
<th>Female</th>
<th>Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonlocalized or generalized</td>
<td>13</td>
<td>57.8</td>
<td>22</td>
<td>11</td>
<td>14/3</td>
</tr>
<tr>
<td>Bifrontal</td>
<td>6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsilateral frontal</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contralateral frontal</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsilateral retro-orbital</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipsilateral frontotemporal</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occipital</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Time relation onset headache to other symptoms**

| Before | 5 |
| During | 15 |
| After | 12 |
| (One uncertain) | |

**Constancy of relationship (if more than one TIA)**

| Constant | 7 |
| Inconstant | 16 |
| Either one TIA or unknown | 10 |
HEADACHE AND TRANSIENT ISCHEMIC ATTACKS

TABLE 3

<table>
<thead>
<tr>
<th>Location of headache</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonlocalized or generalized</td>
<td>5</td>
</tr>
<tr>
<td>Bioccipital</td>
<td>7</td>
</tr>
<tr>
<td>Unilateral occipital</td>
<td>3</td>
</tr>
<tr>
<td>Occipital-nuchal</td>
<td>4</td>
</tr>
<tr>
<td>Nuchal</td>
<td>1</td>
</tr>
<tr>
<td>Bifrontal</td>
<td>2</td>
</tr>
<tr>
<td>Biparietal</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Time relation onset headache to other symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
</tr>
<tr>
<td>During</td>
</tr>
<tr>
<td>After</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Constancy of relationship (if more than one TIA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
</tr>
<tr>
<td>Inconstant</td>
</tr>
<tr>
<td>One TIA or undetermined</td>
</tr>
</tbody>
</table>

The pathophysiology of headache occurring in association with cerebrovascular disease has been comprehensively reviewed. Pain-sensitive structures as well as pain pathways from the dura and cerebral vessels have been well established. Pain arising from supratentorial structures is generally referred frontally, whereas that arising infratentorially is localized to the occipital or nuchal regions. According to Fisher, the basic atherosclerotic process as well as uncomplicated cerebral infarction are painless. Wolff proposed that the headache of carotid thrombosis resulted from inflammation of the carotid wall and that irritation near the bifurcation may reflexly initiate painful vasodilation. In reviewing the premonitory headache of cerebral embolism, Wells attributed the pain to embolic distortion of the arterial wall at the base of the brain which ceased when the embolus dislodged and moved distally. Widespread changes in the arterial walls at the base of the brain was proposed to explain the occasional occurrence of generalized headache in these patients. Referred pain secondary to dural irritation and cervical vertebral compression have also been reported in association with cerebrovascular ischemia. Although the anatomic origins and pain pathways have been established, their mechanisms remain uncertain.

Although our data do not reveal the relative incidence of headache in the carotid and verteobasilar TIA groups, this information, although variable, has been reported by others. In patients with carotid stenosis or occlusion and subsequent infarction, headache occurred premonitorily in from 13.9 to 50%. In Loeb’s series, headache as a prodromal sign of focal carotid ischemia was more common in patients with carotid and middle cerebral occlusion than in those without occlusion. Headache related to verteobasilar insufficiency has been reported in from 10% to 54%. The problems encountered in determining the frequency and significance of headache occurring with cerebrovascular disease have been reviewed. Factors such as memory loss, prior history of headache, aphasia, hypertension and sensory loss which may modify the headache limit the reporting as well as the interpretation of these symptoms.

The localization of headache in our series is comparable to that reported by others, although Fisher also reported two patients who complained of neck discomfort (carotodynia) in the region of the involved carotid. Headache secondary to carotid artery disease has been reported as moderate in degree, non-pulsatile, and generally of brief duration.

In five patients, headache was associated with amaurosis fugax. In three, the pain was localized to the orbit of the affected eye and was described as severe and stabbing in two. In one case, the pain was localized to the ipsilateral temporal region and generalized. Our findings in this regard are contrary to those of Fisher who reported no complaints of headache among 58 patients with monocular blindness.

The association of headache with verteobasilar disease has been emphasized. Williams and Wilson reported headache in 25% of cases with basilar infarction and in 10% with verteobasilar insufficiency. Headache was described as occipital, often unilateral and frequently associated with occipital and neck tenderness. Bradshaw and McQuaid emphasized the occipital localization and commented on the frequent prolonged nature of the headache and accentuation of pain with stooping or straining. Although our findings support the occipital localization and frequent persistent nature of such pain, we have no data regarding precipitating or aggravating factors.

Headache in association with the subclavian steal syndrome is generally regarded as uncommon. Our data reveal six patients in whom this association was present. The headache was occipital or nuchal in four and ranged from mild to severe in intensity. Headache tended to occur with the onset of accompanying neurological symptoms and was generally a constant
feature during recurring attacks. Schott et al.\textsuperscript{14} also recognized the occurrence of headache with the subclavian steal syndrome and postulated a hemodynamic mechanism for the pain.

Conclusions

1. Headache is a prominent symptom in approximately 25\% of patients with TIA and may be the presenting complaint.
2. There are no distinguishing population, arteriographical, or prognostic features among patients whose TIAs are accompanied by headache when compared to those with no headache.
3. Frontal headache is more frequently associated with TIAs in the carotid territory whereas occipital-nuchal headache was more common in vertebrobasilar TIA. Nonlocalized headache occurs frequently in both groups.
4. With TIA in both carotid and vertebrobasilar distributions, headache usually accompanied or followed the TIA and only in a few cases preceded the onset of other neurological symptoms. The character of the pain and its duration were not remarkably different between the two groups.
5. In patients with vertebrobasilar insufficiency, headache was more likely to be a constant feature in cases with more than one TIA when compared to those with symptoms in the carotid distribution.
6. Headache was sometimes reported by patients with amaurosis fugax and subclavian steal syndrome, an association in these two conditions which in the past has been felt to be infrequent.

References

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