Isolated Ophthalmic Migraine in the Differential Diagnosis of Cerebro-Ocular Ischemia

THOMAS R. HEDGES, M.D., AND RICHARD D. LACKMAN, B.S.

SUMMARY  Thirty-three of 129 patients who incurred isolated ophthalmic migraine had monocular attacks of scotomatous visual field loss. Fifteen of 33 patients with monocular attacks had immediate or remote evidence of vascular disease. Four patients had carotid bruits on the same side as the monocular attacks and low ophthalmodynamometer readings. One patient had ischemic optic neuropathy and two had atheromatous disease (advanced stage in one patient). Forty-five percent of the patients with monocular attacks and only 13% of the remaining patients with homonymous attacks had vascular complications. This represents an important finding even in such a small group of patients. It is felt that, whether the vascular problems are trigger mechanisms or coexistence pathology to the migraine-type attack, one should strongly suspect such an association when a patient describes a monocular attack and one should look for a possible vascular explanation other than migraine.

Introduction

VISUAL OBSCURATIONS are a well-recognized prodroma of classic migraine. Isolated episodes similar to the classic fortification scotoma and variations of these visual attacks not accompanied by subsequent vascular headache are also a common disorder.

The phenomenon of isolated ophthalmic migraine was well recognized as early as the nineteenth century by Charcot who termed it “migraine fruste.” In 1936, Johnson described his own attacks of isolated scintillating scotomas. Since then the literature has been summarized by the reviews of Alvarez, Aring, and Hedges, dealing specifically with the incidence and diagnosis of these attacks.

Little attention has been given to distinguishing whether these attacks, which simulate the prodroma of migraine, occur in a monocular or a binocular fashion. This distinction may well be one of importance in properly classifying this phenomenon as to its etiology and eventual prognosis. It is for this reason that this series of 129 patients is presented.

Clinical Material

Placing patients in the category of “isolated ophthalmic migraine” is not, by the nature of the disorder, a precisely quantitative procedure. Relatively objective criteria were used for the proper selection and classification of patients. All of the patients must have fulfilled all of the following requirements in order to be designated as having experienced isolated ophthalmic migraine.

(1) The patient must have experienced a visual obscuration lasting not less than five minutes (though most lasted 15 to 30 minutes). Qualitatively these visual symptoms were fairly uniform in that most resembled the classic fortification scotoma in migraine with or without accompanying scintillations.

(2) These episodes were not associated with subsequent headaches.

(3) The visual symptoms were not accompanied by any other neurological deficits (i.e., motor or sensory loss, etc.).

(4) The attacks must have cleared completely leaving no residual visual deficit.

(5) Patients who were on medications which may produce visual symptoms as known side effects were excluded.

Each patient considered had a carefully performed ophthalmological examination including a detailed history. In addition, radial and carotid pulses, auscultation for bruits, ophthalmodynamometry and visual fields were performed. Few of the patients had complete blood, general physical or neurological studies except where specifically indicated and thus one cannot rule out some possible vascular, hematological or other contribution to the visual symptomatology.

Results

Table 1 shows that 60% of the patients were between 50 and 81 years of age; 53% were women. Seventy-five percent of the patients had homonymous hemianopic scotomas without headache (as in the prodrome of classic migraine). The homonymous scotomas were accompanied by scintillation in 60% and occurred without any scintillation in 36%. An important finding was that 33 of the 129 patients or 25% had specific monocular symptoms. These patients alternately covered one and then the other eye and stated they could be sure the scotomas occurred on one side only.

As in our previous series, most patients (73%), according to their recollection, had less than six attacks per year and 42% had less than four attacks.

Eighty-four patients (65%) had no previous history of migraine though many had a family history of migraine.

Table 2 shows certain aspects of the monocular group which are of primary importance. The average age incidence of the entire group was 52 years old and, of the monocular group, 55 years. Frequency of attacks also was not greatly different between those patients who had predominantly typical scintillating scotomas of a homonymous character and those with monocular attacks (the majority of both groups did not experience more than six attacks per year).

The most striking fact was that 15 of 33 patients with monocular visual attacks (45%) had overt clinical evidence of other vascular complications. Only 12 of the remaining 96 patients (13%) with homonymous hemianopic attacks had evidence of such stigmata on a purely clinical basis (without a thorough vascular or neurological workup).

Table 3 classifies the vascular complications experienced by patients in the monocular group. Four of the patients with monocular symptoms had carotid bruits on the same side as the visual loss. This is of utmost importance despite the small numbers, since monocular amaurosis fugax due...
A vertical jagged border and blurred area in the center which spread to the periphery in the right eye only” (by monocular testing). These attacks of blurring lasted five to ten minutes. He had no other neurological or vascular signs and symptoms until two weeks prior to observation when he suddenly had blurred vision in the right eye. At this time, he had 20/200 vision on the right, an afferent pupillary response deficit on the right as evidenced by pupillary escape to direct light, and a positive swinging light test. The right optic nerve was swollen and visual fields showed a nerve fiber bundle defect on the right (Bjerrum-type scotoma). The diagnosis was ischemic neuropathy. The acute signs resolved but he continued to have 20/70 vision and a small but well-defined central scotoma and optic atrophy.

Also of interest was the fact that two of these 33 patients had had “flashes of light” with a movement spread of the scotoma from center fixation to the periphery. Four patients, including these two, stated the visual phenomenon was always temporal to fixation. In seven patients, the duration was 20 minutes. All of these attributes, save the fact that the visual disturbance was definitely monocular and not in a homonymous scintillating scotoma, could be easily mistaken for a binocular attack unless the patient had been a good observer.

One patient experienced visual disturbances for many years and stated that the 15-minute to 20-minute visual disturbance could occur in both eyes simultaneously or in one eye alone. In each instance the attack was similar; it began temporal to fixation and was accompanied by a pulsating vertical zig-zag line that moved off to the periphery. If the attacks occurred in both eyes, the temporal half of the patient’s vision was affected at fixation as a bitemporal defect through which the patient was able to see enough to read with difficulty. No headache ever followed but some nausea usually accompanied these attacks. The important thing is that three other patients in the monocular group indicated that the scotoma was not completely central but only in the temporal field. These three patients described the duration of their visual attacks as short-lived (five minutes or as long as three hours). All of these symptoms previously described may or may not have a causal relationship to monocular attacks resembling isolated ophthalmic migraine but are certainly an important consideration in reviewing the differential diagnosis of “benign” attacks of isolated ophthalmic migraine and in distinguishing them from incipient stroke symptoms.

<table>
<thead>
<tr>
<th>Age range (years) (average: 55 years)</th>
<th>No. pts.</th>
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<tr>
<td>&lt; 30</td>
<td>2</td>
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<tr>
<td>30-50</td>
<td>10</td>
</tr>
<tr>
<td>51-70</td>
<td>21</td>
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<tr>
<td>Total</td>
<td>33</td>
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<th>Frequency</th>
<th>No. pts.</th>
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<tr>
<td>&lt; 4 (total)</td>
<td>16</td>
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<tr>
<td>&lt; 6 per year</td>
<td>6</td>
</tr>
<tr>
<td>&gt; 6 per year</td>
<td>11</td>
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<th>Vascular complications</th>
<th>No. pts.</th>
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<tr>
<td>Monocular group</td>
<td>15 of 33 (45%)</td>
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<td>Homonymous hemianopia group</td>
<td>12 of 96 (13%)</td>
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*Homonymous hemianopia.
Discussion

A majority of the total group had typical homonymous hemianopic scotomas and therefore they were cortical in origin (75%). It is this group that is relatively easy to diagnose; they have the posterior cerebral artery and calcarine cortex as their anatomical location and, in almost all instances, have a benign prognosis. The monocarotid group, on the other hand, were unusual in that they occasionally had a longer and, even more often, a shorter duration than the usual 20-minute attack of typical homonymous fortification scotoma. An important observation was that they had more frequent vascular complications.

Age, Sex and Previous Migraine History

Vascular complications occurred in 52% of the monocarotid group as compared with 12% in the remaining patients who had typical homonymous attacks. One might have thought this was due to age factors. However, the average age for the total group was 54 years, for the monocarotid group 55 years, and for the 11 patients with vascular complications 53 years. Thus, age would not appear as important as one might have expected. Their sex distribution and the incidence of previous classic or common migraine were the same as in the group as a whole. Two-thirds of the patients had no previous history of migraine.

Carotid Bruit Patients

Three women had carotid bruits, and their ages were 54, 55 and 66. No patient as yet has had a carotid or brachial arteriogram in order to establish a diagnosis of carotid stenosis. All had significantly lower diastolic blood pressure readings, however, the average age for the total group was 54 years, for the monocarotid group 55 years, and for the 11 patients with vascular complications 53 years. Thus, age would not appear as important as one might have expected. Their sex distribution and the incidence of previous classic or common migraine were the same as in the group as a whole. Two-thirds of the patients had no previous history of migraine.

Perhaps in some patients who never had a family history of or an attack of migraine, vascular insufficiency is a precipitating or trigger mechanism for an attack which is really a transient ischemic episode rather than a purely migraine phenomenon. Alvarez² feels that cerebral arteriosclerosis contributes to the clinical picture where migraine-like attacks are really "little strokes." He stated further that his own scotomas, which were not typical in his older years, may well have been due to arterial spasm or small thrombosis of cerebral arteries.

McDonald and Sanders⁹ described another case of migraine complicated by ischemic optic neuropathy, but this patient had a typical migraine attack and not one simulating isolated ophthalmic migraine as shown here. They stated: "There is ample clinical evidence that during an attack of migraine there may be transient ischemia of the visual system at a retinal, chiasmal or cortical level and lasting symptoms may occasionally arise from involvement at any of these levels." This brings us to the anatomy of these attacks. The above statement by McDonald and Sanders can be contested only as regards the chiasm. The vulnerability of the blood supply to the optic nerve and retina as well as the posterior cerebral calcarine circulation is well recognized. The optic disk area, as pointed out by these authors, also is very vulnerable to vascular insufficiency. The chiasmal area, however, is not so prone to ischemia. It is noted for its invulnerability to vascular occlusive disease because of its rich and diverse blood supply. When faced with patients in this monocarotid group who have temporal scotomatous attacks (even the one who said the attacks occurred bilaterally on rare occasions), one is forced to think anatomically of the nerve and especially the anterior optic nerve. Here it would be perfectly plausible to get an attack of amaurosis with a subjective field defect temporal to fixation which is in the form of a central scotoma or nerve fiber bundle defect. These patients with flashes of light as well and a "spread of spectral march" in one eye similar to the cortical attack patients are more enigmatic. One cannot explain these attacks on a cytoarchitectural basis as one is able to do in the visual cortex. Attacks of bright lights are frequently observed by patients with retinal disease. These are usually stationary in one field and do not progress in an orderly fashion from center to periphery as described by some of the patients in this study.

References

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Isolated ophthalmic migraine in the differential diagnosis of cerebro-ocular ischemia.

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*Stroke*. 1976;7:379-381
doi: 10.1161/01.STR.7.4.379

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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