

# Classic Migraine With Cerebral Cortical Infarction Causing Permanent Hemianopia

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**ABSTRACT:** A patient with typical classic migraine, including clear-cut visual auras, who had been followed up clinically for more than 15 years developed permanent right homonymous hemianopia. The underlying cause of this clinical syndrome was established by computerized axial tomography as vascular infarction or ischemia, involving the contralateral visual cortex.

IN THIS AGE of nuclear power, moon "walks," heart transplants, and synthetic genes, we cannot yet fully understand or prevent classic migraine (CM), but recent scientific advances, including computerized axial tomography (CAT), offer great promise.<sup>1-5</sup>

Accurate classification of headache and migraine seems almost impossible at present because of uncertain etiology<sup>6</sup> and a host of confusing diagnostic types and terms. However, a very comprehensive study of the problem has been provided by the Ad Hoc Committee on Headache,<sup>7</sup> and their definitions are followed here. The prodromal warning aura of impending CM is limited to clear-cut visual prodromal auras such as scintillating scotomas (SS). CM is an episodic disorder characterized by upper headache, beginning without obvious cause, often in a healthy-appearing person, and preceded always by a clear-cut visual warning aura such as SS, then followed by prolonged headache, accompanied usually by anorexia and nausea with occasional emesis, but ending spontaneously after many hours or a few days, almost always without permanent sequelae or visual deficit.

The history of migraine has been traced far into antiquity by Alvarez,<sup>8</sup> who found that the descriptive term "scotoma scintillans" had been used as early as 100 AD. He also predicted that prolonged recurrent CM might lead eventually to permanent visual deficit or even blindness. The following case report offers good support for this and seems to indicate that the underlying cause of CM is in the vasculature of the visual cortex of the cerebrum.

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## CASE REPORT

A white man with no complaint except occasional attacks of prolonged "sick" headache or migraine, was first examined at about age 45 years. A family history of recurrent "sick" headache or migraine was clearly described, involving each parent and two of four siblings. All use of drugs, including tobacco, coffee, and alcohol, was firmly denied. Physical examination revealed no abnormality of note, and the patient's general health appeared to be excellent. Height was 175 cm (70 inches), weight was 70 kg (154 lb), and blood pressure, 130/70 mm Hg. Peripheral pulses and reflexes were present and unchanged. No cardiac murmurs or vascular bruits were audible. Roentgenographic studies and clinical laboratory findings were well within normal limits.

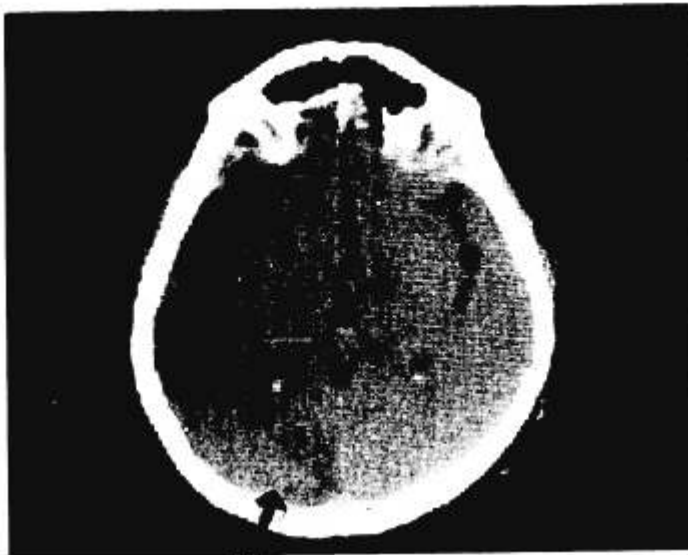
The patient recalled many headaches and probable common migraine attacks as early as 6 or 7 years of age but at 16½ years the onset of real CM was heralded by a sudden, alarming attack of dazzling semiblindness that was later identified as an aura of CM. This first warning aura (and almost all later auras) cleared spontaneously within an hour, without permanent sequelae. The ensuing prolonged upper headache or CM was usually bilateral and began not long after the aura; it was accompanied by slight anorexia and prolonged mild nausea with occasional slight terminal emesis. The duration of the entire episode was about two days.

During the patient's most active years, from about 17 to 40, he recalled relatively few attacks, but as vigorous athletic activity decreased, the CM increased.

At about age 45, one of the usual CM episodes was complicated by severe prolonged diplopia and vertigo, for the first time necessitating hospitalization. All symptoms cleared spontaneously. Examination, including special neurologic studies and cerebrospinal fluid examinations, revealed no abnormality, and roentgenographic and clinical laboratory studies also were well within normal range. The patient returned to his usual occupation, and the severe diplopia with vertigo has never recurred.

During the years from about age 40 to 50, the patient had numerous episodes of recurrent typical CM which always cleared spontaneously, leaving no permanent sequelae. Near the end of this period, while he was driving cross-country, an attack of CM began as usual with an aura of SS, but this time the aura persisted unusually long and was quite dazzling throughout. After several hours it merged directly into localized "spot" blindness that was diagnosed by ophthalmologists as probable incomplete homonymous hemianopia (HH). This limited or partial HH caused loss of vision in a very small arc of each mid-right visual field, and it proved to be permanent but was never an obvious visual handicap.

This visual condition persisted unchanged during the ensuing year until the patient made a second cross-country drive, when an attack of CM began as usual with prodromal dazzling SS, but this aura became very dazzling and was far more prolonged than any



CAT scan shows cerebral cortical left occipital lobe infarct which caused right homonymous hemianopia.

prior attack. After many hours it finally merged directly into semi-blindness that was diagnosed promptly by ophthalmologists as essentially complete *right* HH. These two similar HH attacks seemed to involve the same area of each mid-right visual field but the second zone of HH was far the larger and it completely covered and surrounded the first. It caused loss of vision in virtually the entire right half of each visual field (excepting the small macula). Computed tomography (CT) scan revealed a typical thromboinfarction in the left posterior occipital (visual) cortex, near the left calcarine fissure, causing right (contralateral) HH (Figure).

Repeated ophthalmologic studies during the ensuing week showed no improvement, and a second CT scan confirmed the first and demonstrated, even more clearly, the same type of vascular cerebral cortical lesion. These findings documented disease underlying this syndrome as cerebrovascular failure.

During the ensuing three years, repeated examinations revealed no improvement in the patient's vision. The SS attacks continued to recur almost as before, except that the chief site and side of involvement seemed to have changed or moved from the virtually blind right side to the relatively normal left side, ie, the mid-left visual field of each eye. Occasionally, however, the aura and the arc of dazzling blindness has included also the central macula lutea, the loss of which caused virtually total temporary bilateral blindness. This seemed to warn of possible future permanent bilateral blindness or double HH, as described by Harrington.<sup>9</sup>

#### DISCUSSION

Ophthalmologists, neurologists, and pathologists have long known that brain operations or trauma involving certain areas of the cerebral visual cortex may cause permanent (contralateral) HH (or hemibindness) and that certain kinds of migraine may involve these same areas of the visual cortex.<sup>9</sup> Conclusive evidence for these concepts, however, has been difficult to

fully establish in nontraumatic cases without recourse to necropsy studies. Now, however, this problem seems to have been solved by use of the noninvasive CAT scanner, which is reportedly 100 times more sensitive than the conventional roentgenogram.<sup>10</sup>

Migraine is often regarded as a minor malady and it usually proves to be benign, despite a few reports of sudden death occurring during a CM attack,<sup>11</sup> notably one described by Peters in 1934.<sup>12</sup>

Since the advent of modern oral contraceptive drug therapy with its reports of vascular damage and disasters, including "stroke," it seems logical now also to scrutinize migraine therapy, since oral migraine therapy includes many nonprescription drugs that are easily obtained, are often ingested in excessive doses without proper advice or supervision, and are taken to relieve innumerable complaints in addition to all kinds of headaches as well as migraine. The total number of such drug-related untoward reactions, accidents, or disasters is no doubt a public health hazard of considerable magnitude.

#### CONCLUSION

This case seems to establish the cause of classic migraine, with its scintillating scotoma and homonymous hemianopia, to be localized to cerebrovascular ischemia or infarction involving the contralateral visual cortex of the cerebrum. Final documentation of the cause was established by repeated computerized axial tomography scans.

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