Chapter 2

The Basketball Player with Visual Disturbance

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Case History

A 30-year-old electrical engineer presented with a history of moderate unilateral headaches extending back to late adolescence. In college, he had experienced occasional spontaneous headaches that were generally preceded by a visual disturbance that appeared without warning, just lateral to his point of central visual fixation. This visual disturbance tended to expand with time to involve half of the visual field in both eyes. Along the expanding margin, a geometric shimmering pattern was present that persisted when he closed his eyes. In the wake of this expanding prismatic rim was an area devoid of any image. The visual disturbance resolved after 30 to 40 minutes but was followed by the gradual onset of a moderate throbbing frontal headache associated with light and sound sensitivity and, only rarely, nausea. The headaches generally lasted a few hours before resolving spontaneously or after treatment with an over-the-counter analgesic. During his early twenties, the episodes began to occur when he took part in certain sports for more than 30 minutes. He was able to jog or run for long periods of time without the induction of a headache episode. However, his participation in sports such as basketball, which involved frequent stopping and starting or running for short bursts, almost always resulted in the induction of the visual disturbance and headache. The episodes usually followed the sport by about 30 to 60 minutes. If he continued to play after its onset, the headache intensified. On presentation, almost all of his headaches followed basketball playing. The patient presented for evaluation because of his increasing inability to take part in sports that he had always enjoyed.

Questions on the Case

Please read the questions, try to answer them, and reflect on your answers before reading the author's discussion.

- What is the most likely diagnosis?
- Does induction of headache by exertion increase this patient's risk of having an aneurysm or other intracranial abnormality?
- Does the visual disturbance represent a type of seizure or a manifestation of ischemia?
- How would you manage this patient's headaches, and what specific therapies would you suggest? If your initial therapies are not successful, then what would you plan for the future?

Case Discussion

The most likely diagnosis in this case is migraine with visual aura. The patient's description of a gradually expanding prismatic visual disturbance that affects a hemifield of vision in both eyes is quite classical for migraine aura. The subsequent headache of moderate intensity, which persisted for hours and was associated with light and sound sensitivity and nausea, is also consistent with migraine.

The fact that his attacks are initiated by exertion does not in and of itself put him at higher risk for an intracranial abnormality. Certain types of exercise- or exertion-induced headache, such as headache induced by sexual activity, have been linked to sentinel leaks of aneurysms in some case series. The headaches found to be associated with aneurysms or subarachnoid hemorrhage in these series had a sudden explosive onset and other characteris-

tics that would suggest subarachnoid hemorrhage. It is still a matter of some debate as to whether the history of induction with exercise should alone automatically dictate the need for neuroimaging with magnetic resonance angiography (MRA) to screen for a vascular abnormality. However, many experienced clinicians would obtain an MRI/MRA of the brain to eliminate the possibility of a vascular lesion. It is reassuring that this patient had been having these stereotypical headaches for almost a decade without any other evidence of a bleed or aneurysm. Similarly, the presence of the aura and the headache's gradual onset also argue against aneurysm as a cause. In fact, this patient had completely normal cerebral vasculature.

The clinical characteristics of the patient's visual disturbance make an underlying seizure unlikely. The long duration (30 to 40 minutes) and the bimodal progression from positive visual symptoms (prismatic, geometric visual hallucination) to negative ones (scotomata) are not suggestive of seizures. To investigate the possibility of an ischemic cause, this patient was studied during his visual auras using functional magnetic resonance imaging (fMRI). Perfusion-weighted imaging obtained in the minutes after aura showed decreases in relative cerebral blood flow, averaging about 30% in the occipital cortex contralateral to the affected visual field when compared to that on the opposite side of the brain. A 30% reduction is well above the ischemic threshold. Furthermore, blood oxygen level dependent imaging obtained during the aura showed a *decrease* in levels of deoxyhemoglobin areas of occipital cortex corresponding to the visual field abnormalities. If ischemia were present, one would expect an increase in deoxyhemoglobin levels as more oxygen is extracted from hemoglobin during the oxygen shortfall. Interestingly, the first part of this patient's brain to show changes on fMRI during the aura was area V3a, an area of visual association cortex rather than primary visual cortex.

In managing this patient, it was important to remember that almost all of his headaches at the time he presented were induced by physical exercise. He was very active and reluctant to take any medication that could cause sedating side effects. It was initially suggested that he pretreat with the nonsteroidal anti-inflammatory drug (NSAID) ketoprofen 75 mg, about 1 hour prior anticipated exercise. He played in two different basketball leagues during the season and, between games and practices, took ketoprofen about 3 to 5 days per week, with a reliably good result. In those games in which he pretreated, he did not develop the aura or the headache. The advantages of this approach were that he did not have to take a medication on days that he did not play basketball, and a lack of sedating side effects. The disadvantage was the possibility of gastrointestinal irritation if he needed to take the medication several days per week. Frequent use of an NSAID over a long period also carries a risk of renal injury. If the patient had been intolerant of the NSAID or had found it to be ineffective, then other possibilities may have included a beta-blocker (eg, atenolol or propranolol), a calcium channel blocker (eg, verapamil), a tricyclic antidepressant (eg, amitriptyline), or an anticonvulsant (eg, gabapentin or topiramate). The beta-blockers and calcium channel blockers are without the side effect of sedation, but they might have lowered the patient's blood pressure and possibly blunted his athletic performance. The tricyclics and anticonvulsants might be effective but tend to have sedation as a side effect. In this patient, sedation was particularly undesirable.

Case Summary

- This is a typical case of migraine with aura.
- The aura can be differentiated from epilepsy by the clinical features of the episodes preceding headaches.
- Study of the episodes of visual aura in this patient suggests that his symptoms are not based on ischemia.
- The induction of headache with certain types of exercise is associated with an increased risk of intracranial vascular lesions.
- If migraine attacks are induced solely by a predictable trigger, and trigger avoidance is not an attractive option, then short-term prophylaxis may be a viable alternative.
- The choice of a prophylactic agent may in some cases be determined by the side-effect profile of the potential therapeutic agents.

Selected Readings

Cutrer FM, Boes C. Cough, exertional and sex headache. Neurol Clin North Am 2004;22:133–49.

Dodick DW. Acute and prophylactic management of migraine. Clin Cornerstone 2001;4:36–52.

Hadjikhani N, Sanchez Del Rio M, Wu O, et al. Mechanisms of migraine aura revealed by functional MRI in human visual cortex. Proc Natl Acad Sci USA 2001;98:4687–92.

Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias, and facial pain. Cephalalgia 2004;24 Suppl 1:1–160.

Editorial Comments

The case described by Dr. Cutrer is famous, as the functional MRI studies on the patient have been published. They show that human aura is caused by cortical spreading depression, which is in fact cortical spreading activation, followed by a postictal neuronal quiescence, which as Dr. Cutrer points out is not ischemic. Aura is neuronal, not vascular.

Furthermore, unlike migraine without aura, which must be diagnosed on characteristics of the headache, migraine with aura is more readily diagnosed on the neurologic symptomology that accompanies the headache, usually before, and is most commonly visual in nature. The headache intensity in migraine with aura is often mild. This case illustrates the complexity of the visual aura and the fact that sophisticated imaging techniques allow a better understanding of the putative pathophysiology, and the knowledge that the vascular changes for the most part are subischemic. Also, some practical comments on treatment options in a young active person are welcomed. Some physicians would also consider indomethacin 1 hour prior to exercise if ketoprofen were not helpful. If gastric irritation occurs, then a protein pump inhibitor could be given daily. This case is very instructive in a scientific and practical fashion.

Final diagnosis:

Migraine with typical aura