Exercise-Induced Homonymous Quadranopsia after Head Trauma

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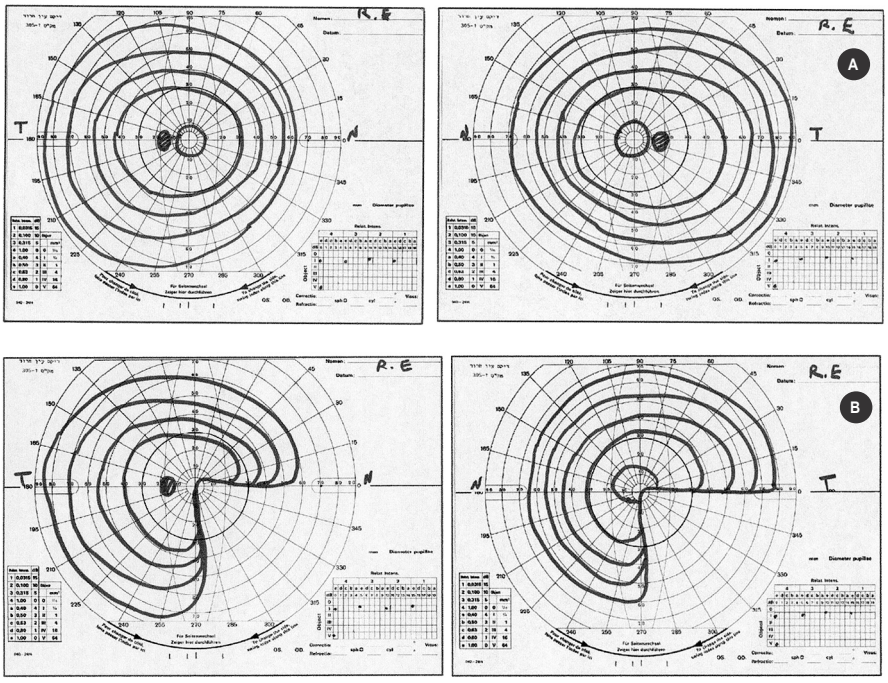
Exercise-induced vision disturbances are uncommon. Most reports in the literature describe patients with either a vascular or demyelinating disorder. Uhthoff's phenomenon is a classic example, commonly described in patients with multiple sclerosis. In 1971, Fisher [1] reported a 56 year old man who experienced episodes of bilateral altitudinal visual loss induced by running and mowing the lawn. The patient was found to have bilateral occlusion of the internal carotid arteries. An additional study describing exercise-induced visual loss was conducted by Shah et al. [2], in which three young patients with congenital or juvenile-onset glaucoma presented with visual loss occurring during exercise.

Reports of this phenomenon in young healthy people are rare. In 1989, Imes and Hoyt [3] reported six cases of healthy young adults, without a history of migraine, who experienced a variety of transient visual events precipitated by exercise. To the best of our knowledge, there are no reports in the literature regarding visual field defects induced by exercise in otherwise healthy adults. We describe a case of a 27 year old healthy young man, without a history of migraine, who experienced recurrent visual field defects precipitated by exercise since an event of head trauma 7 years earlier.

Patient Description

A 27 year old apparently healthy male student presented with recurrent similar visual field defects induced by physical exertion such as running or playing basketball. The disturbances lasted 60–90 minutes and were unassociated with headache, although if physical activity was not stopped at once transient dizziness appeared for approximately 2 hours. The patient had no history of migraine, recurrent headaches or somnambulism. He had, however, experienced a brain concussion 7 years earlier during his military service, due to a blunt trauma. The trauma, caused by a 50 kg metal object, was characterized in part by a loss of the right homonymous visual field lasting 36 hours, severe headaches and nausea. Electroencephalogram was normal. Computed tomography and magnetic resonance imaging were performed at this time and were interpreted as normal. The patient's visual episodes began 2–3 months post-trauma and subsequently occurred during/after almost every aerobic exertion lasting more than 5–10 minutes.



[A] Goldmann visual fields in rest. **[B]** Goldmann visual fields 15 minutes after exercise.

On examination, the patient's visual acuity was 6/6 in both eyes, color vision (Ishihara color plates) was normal (15/15), eye movements were full, and he had normal intraocular pressure in both eyes. His pupils were oval and normally reactive to direct and consensual light stimulation. Fundus examination revealed normal optic discs with no arterial or venous constriction. Visual fields were normal by kinetic perimetry [Figure A]. After 15 minutes of exercise (briskly climbing 20–25 floors), he reported visual loss to the right in both eyes. A repeat neuro-ophthalmologic examination was performed and was unchanged except that perimetry revealed a right inferior homonymous quadrantanopia (or incomplete homonymous hemianopia,

denser below) [Figure B]. This test method was verified again on different occasions with identical results.

The patient thereafter noted a gradual resolution of the visual field defect, starting from the periphery towards the center. A repeat perimetry 2 hours later was normal. Blood pressure and heart rate before and during exertion were within normal limits. General blood chemistry profile, blood gases and electrolytes, electrocardiogram and repeat MRI were all normal. In addition, a Technetium-99m HMPAO brain SPECT was performed. The injection was given after 15–20 minutes of physical exertion on a treadmill, after the patient reported the beginning of his regular visual defect "symptomology." The results showed low absorption, with a patchy appearance, and no substantial perfusion deficit in any specific cortical region.

Comments

The most common exercise-induced visual event is Uhthoff's phenomenon. First described in 1890, it occurs primarily in patients with multiple sclerosis, especially those with optic neuritis. In such patients, small increases in body temperature caused by exercise, hot baths, showers, etc., cause the development of new neurologic symptoms such as visual loss, or worsening of preexisting symptoms, probably related in most cases to interrupted conduction in partially demyelinated axons. Affected patients often lose transient vision in one or both eyes during exercise or when overheated.

It is known that physical exertion can aggravate a migraine headache. However, the relationship between exertion and the migraine aura is unknown [4]. The usual duration of a migraine aura is less than an hour, although it can last up to a week. Migraine aura is thought to result from cortical spreading depression. It is unclear, however, if additional mechanisms, such as ischemia, contribute to prolonged aura. Indeed, Razavi et al. [4] claim that ischemia is not a major contributory factor. Our patient did not fulfill the criteria for migraine with an aura or migraine without headache, as the visual field loss occurred only and always immediately after exertion. In addition, he had never suffered from migraines or recurrent headaches of any sort. We presume that this event was due to the past head trauma.

Exertion is associated with hyperventilation and hypocapnea, both of which are known to cause cerebral vasoconstriction. In addition, hyperventilation can cause respiratory alkalosis and hypomagnesemia. Because hypomagnesemia can cause vasoconstriction, the transient hypomagnesemia induced by hyperventilation, combined with primary magnesium deficiency seen in migraine sufferers, may exaggerate the vascular response [5]. However, no hypomagnesemia was found in our patient (2 mg/dl).

We presume that exertion-induced vasoconstriction of cerebral vessels may be due to hypersensitivity to certain vasoconstrictors released during physical exercise or by an inappropriate high release of such mediators as catecholamine or other neuropeptides. We believe that the brain concussion caused the vasculature of the occipital cortex to become more susceptible to physical exertion, resulting in transient vasospasm. It is important that the clinician be aware of the existence of this rare phenomenon and that he or she accurately diagnose this condition, which is definitely not due to mental illness or structural lesion. It should be explained to the patient that this is a benign but well-known phenomenon.

To our knowledge, this is the first report of an exercise-induced, transient homonymous visual field defect in an otherwise healthy person.

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