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Movement Phosphenes in Optic Neuritis

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Abstract

Patients with optic neuritis may note bright-colored flashing lights upon entry into a dark room, and these movement phosphenes may be aggravated by horizontal eye movements. Three patients with this phenomenon are described. It may be an irritative symptom in the optic nerve analogous to Lhermitte's sign in the spinal cord. The differential diagnosis of "flashing lights" is presented.

In 1976, Davis et al. noted an association between eye movement-induced positive visual phenomena (movement phosphenes) and optic neuritis.[1] Since that report, no further mention of this interesting phenomenon has appeared in the literature. It is the purpose of this report to present three additional cases involving movement phosphenes in optic neuritis, review the differential diagnosis of "flashing lights," and suggest possible mechanisms for their appearance.

Methods and Materials

Patients seen within the last year with optic neuritis were asked whether they had noted "flashing lights" prior to, during, or subsequent to the onset of their disease. For the purpose of this study, the patients were contacted by telephone in an effort to update and confirm the information contained in our records. Twelve patients with optic neuritis were seen, and three reported "light flashes" during or after the onset of optic neuritis.

Case 1

A 25-year-old woman complaining of decreased visual acuity in the right eye first noted an acute loss of vision on the right side in 1977. The visual acuity was light perception in the right eye and 20/20 in the left eye. The acuity in the right eye improved over several weeks to 20/30. There was a recurrent loss of vision in the right eye in August 1978 with the visual acuity at that time dropping to 20/200 in the right eye. Visual acuity in the right eye returned to 20/40 in September 1978. In February 1978, the patient had visual-evoked potentials, which revealed a conduction delay on the right side only.

The patient was seen in consultation on April 2, 1980, because of loss of visual acuity in the right eye associated with pain on eye movement. She had noted decreased vision in both eyes during strenuous exercise for the past year. Visual acuity was 20/70 in the right eye and 20/30 in the left eye. No afferent pupillary defect was seen. On visual field testing, bilateral central scotomas were noted. Mild optic atrophy was seen on the right side, but the left disc appeared normal. It was felt that the patient had bilateral optic neuritis clinically evident on the right side and relatively asymptomatic on the left. A neurologic examination was completely negative. A skull series with optic canal views was found to be within normal limits, and computed tomography with a fourth generation scanner revealed thinned and attenuated optic nerves bilaterally. A spinal tap, including protein and IgG levels, was normal. The visual acuity gradually improved to 20/40 in the right eye but remained at 20/30 in the left eye. In the following months, the patient began noting bilateral attacks of complete amaurosis fugax. Most of these amaurotic attacks lasted only minutes, but occasionally would last for several hours.

Neuro-ophthalmologic examination in February 1981 revealed a corrected visual acuity of 20/30 in both eyes. There was bilateral peripheral visual field constriction in conjunction with bilateral central and arcuate scotomas. There was diffuse drop-out of the nerve fiber layer in both eyes. The patient reported for the first time that she noted colored flashing lights in both eyes when in a dark room during the past several months. They were frequently associated with eye movement. She described the flashing lights as resembling images seen when "looking through a kaleidoscope," start-

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ing in the central field and moving to the periphery. These were not associated with headache, nausea, or vomiting. The episodes of flashing light usually lasted less than 1 minute. Repeated neurologic examination was entirely normal except for the ophthalmologic findings. Subsequently she complained of marked fatigue, which was exacerbated by taking a hot shower.

Case 2

A 36-year-old white woman presented to her local ophthalmologist with a complaint of blurred vision in her left eye. The blurred vision had come on acutely 1 week prior to this examination. The patient had noted blurriness and wavy lines in the lower visual field of the left eye which progressed centrally, making it difficult to perceive colors or letters. She also had noted the simultaneous onset of a dull pain in her left temple, numbness of her left forehead, left cheek, and in back of her left ear. Her visual acuity at that time was 20/20 in the right eye and 20/40 in the left eye. The pupils were equal and reacted normally to light and a near target. A left afferent pupillary defect was apparent. There was a slight decrease in adduction of both eyes and slowing of saccades on adduction, consistent with a mild bilateral internuclear ophthalmoplegia. Fundus examination was normal. Visual field testing was normal in the right eye but showed a markedly constricted visual field in the left eye with a dense inferior arcuate scotoma. There was a decreased corneal reflex in the left eye. There was decreased sensitivity to pinprick on the left forehead and left cheek. Neurological examination was otherwise normal. The patient was felt to have retrobulbar neuritis and probably demyelinating disease. Three days later she was admitted to the hospital because of persistent pain and decreased vision in her left eye. Blood chemistries were within normal limits. Electroencephalogram remained normal during intermittent photic stimulation and hyperventilation. Skull x-rays with views of the optic canal were normal. Cerebrospinal fluid analysis was normal except for a total protein of 50 mg/dl (normal = 15–45 mg/dl) and an IgG component of 14.5% (normal < 14%). The patient was started on a 10-day course of ACTH, and her pain subsided within several days. She was discharged with a visual acuity of 20/40 in the left eye.

One month later she noted left eye pain but no further facial hypesthesias. Her ophthalmologic examination was unchanged. She was started on a short course of high-dose prednisone (100 mg tapered to 10 mg in 18 days). Two weeks later the patient noted improved vision, and her visual acuity in the left eye at that time measured 20/25.

Two months later, on July 22, 1980, the patient was seen in consultation at the Albany Medical College because of increased difficulty in judging distances and in viewing moving objects. It was at this time that she first complained of flashing lights when she entered a dark room. The lights were always associated with horizontal eye movements and were described by the patient as diffuse, flashing, colored lights occupying the whole visual field of the left eye. On careful questioning, it was discovered that this phenomenon had been present 1 week after the onset of the initial decrease in vision. Examination revealed a corrected visual acuity of 20/20 in both eyes. There was a left afferent pupillary defect. Visual field testing revealed an enlarged blind spot in the left eye. Ophthalmoscopy showed slight optic atrophy and thinning of the left inferior temporal nerve fiber layer in the left eye. Visual-evoked potentials revealed a marked conduction delay in the left eye. The right eye was normal. The impression at that time was retrobulbar neuritis with good recovery. The difficulty experienced with moving objects was thought to be due to the conduction delay in the left eye, causing a distortion of objects in motion, *viz.*, the Pulfrich phenomenon. The eye movement-induced "flashing lights" were thought to be movement phosphenes associated with optic neuritis.

Six months later the patient reported intermittent eye pain and blurred vision when she was fatigued. Her neurologic examination has otherwise remained normal.

Case 3

A 30-year-old man complained of experiencing bilateral orbital pain for 10 days, the left side more painful than the right side. The pain was constant and aggravated by eye movement, especially extreme left gaze. Horizontal diplopia was also noted on extreme left gaze. There was no complaint of visual blurring or visual loss.

On examination on January 15, 1981, the visual acuity was 20/20 in each eye. There was no conjunctival injection or proptosis. The pupils were normal, and no afferent defect was noted. There was a slight decrease in abduction of the left eye. The remainder of the ophthalmologic examination was normal, including visual field testing with a tangent screen and Goldmann perimeter. Neurologic examination was also normal.

Complete blood count and sedimentation rate were normal. Roentgenograms of the skull and computed tomography of the orbits, plain and enhanced, were normal.

Three days after the initial examination the patient noted central blurring of the left eye. Visual acuity was noted to be 20/15 in the right eye and 20/20-2 in the left eye. He experienced showers of

flashing white lights upon entering a dark room, which were increased by eye movement. Visual-evoked potentials revealed borderline latencies in both eyes, and a diagnosis of retrobulbar neuritis was made. The "light flashes" diminished as the patient's visual acuity gradually returned to normal over the ensuing 6 weeks.

Discussion

Movement phosphenes were noted in association with optic neuritis in all three patients, although they were not reported during the initial attacks of the disease. All three patients noted the movement phosphenes only when they were in a dark room. In addition, all three noted a reproducible exacerbation of their phosphenes on eye movement. There was no apparent relationship between the appearance of movement phosphenes and the degree of visual dysfunction during the acute phase of optic neuritis. All three patients had a significant return of visual function, which indicates to us that movement phosphenes are not an ominous sign.

Our findings are consistent with those reported by Davis et al.[1] They reviewed nine patients who complained of movement phosphenes and carried a diagnosis of optic neuritis and/or multiple sclerosis. Their patients were similar to ours in that light flashes were usually accentuated by horizontal eye movement in a dark room. In their series, six patients noted movement phosphenes during the acute phase of the optic neuritis, while two patients reported them subsequent to the acute phase. One patient reported movement phosphenes during the active phase of multiple sclerosis, although there was no definitive clinical evidence of optic neuritis at the time.

Phosphenes produced by sudden movements of the eyes (movement phosphenes) were described as early as 1819 by Purkinje.[2] Since then, movement phosphenes have been noted to occur in individuals with normal vision when the eye is placed at the extreme limits of horizontal, vertical,[3] or convergent gaze.[4]

Davis et al. restated that movement phosphenes may be the visual equivalent of Lhermitte's sign. Patients with this sign complain of shock-like paresthesias in the trunk and/or extremities after rapid flexion of the neck. It is seen in association with multiple sclerosis, although the exact etiology remains unclear. Davis et al. postulated that Lhermitte's sign might be produced by the mechanical deformation and firing of nerve axons that had become hyperexcitable secondary to demyelination. Similarly, movement phosphenes may be the result of eye movement-induced mechanical deformation and firing of an optic nerve that had become hyperexcitable secondary to demyelination.

Lhermitte's sign is also seen in association with spinal cord compression syndromes, e.g., cervical spondylosis.[5] Flexion of the neck in the presence of cervical canal stenosis compromises the cord substances by direct mechanical compression, producing the characteristic paresthesias of Lhermitte's sign. Whether demyelination plays a role in the pathogenesis of Lhermitte's sign in this clinical setting is not known. Regardless, mechanical deformation of nerve bundles appears to be the common denominator in the production of Lhermitte's sign and its ocular equivalent, movement phosphenes.

The differential diagnosis one considers when a patient complains of flashing lights commonly includes detachment of the retina,[6] posterior vitreous detachment with vitreoretinal traction,[7] the scintillating scotoma of migraine,[8] and blunt force or deep pressure applied to the eyeball.[4] Less common causes of flashing lights include the appearance of scintillating scotoma without migraine,[9] arteriovenous malformation of the occipital lobe,[10] lesions of the parietotemporal region,[11] and retinal microemboli.[9] Ophthalmologists should include optic neuritis in their differential diagnosis of "flashing lights," especially in the clinical setting discussed.

Summary

Movement phosphenes were noted in three of 12 patients who presented within the last year with optic neuritis. The movement phosphenes were described as bright, colored, flashing lights, which occurred only upon entry into a dark room and were frequently aggravated by horizontal eye movement. Movement phosphenes are a sign of optic neuritis and should be considered in the differential diagnosis of "flashing lights."

(See editorial comments on page 290.)

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