Bilateral Loss of Vision in Bright Light

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We describe four patients with episodic bilateral vision impairment related exclusively to light exposure. Each had angiographically documented bilateral high-grade stenosis or occlusion of the internal carotid arteries. This phenomenon appears to be related to bilateral simultaneous retinal ischemia delaying regeneration of visual pigments in the pigment epithelial layer. It must be distinguished from bilateral occipital lobe ischemia caused by vertebrobasilar system disease. (Stroke 1989;20:554–558)

The phenomenon of unilateral loss of vision in bright light was initially reported as a manifestation of carotid arterial occlusive disease by Furlan et al. These authors described five such patients who also had decreased retinal artery pressure on the side of the vision loss and arteriographic evidence of high-grade stenosis or occlusion of the ipsilateral internal carotid artery.

Subsequently, Donnan et al. studied four cases of severe carotid occlusive disease associated with ipsilateral blurring of vision on exposure to bright light. In these patients, attenuation of the visual evoked response immediately after exposure to bright light was found (this phenomenon was not seen in control patients, except in one in whom a small decrement of evoked response occurred). The attenuation of the visual evoked response was thought to represent a change in the function of the macular zone, presumably because of a decrease in local retinal blood flow that resulted in inadequate regeneration of retinal pigments.

We describe four patients with bilateral impairment of vision upon light exposure and angiographically documented bilateral high-grade stenosis or occlusion of the internal carotid arteries.

Case Reports

Case I

A 61-year-old man experienced two episodes of generalized blurring of vision, light-headedness, and diaphoresis while standing, lasting approximately 20 minutes and unassociated with exposure to bright light. A cerebral arteriogram made in August 1984 showed a large ulcerated plaque at the right carotid artery bifurcation without significant narrowing and mild to moderate stenosis of the left proximal internal carotid artery. Bilateral carotid endarterectomies were performed elsewhere in August and September 1984, without complications.

In February 1987, he suddenly experienced left arm and leg weakness lasting 1–2 days. Computed tomography (CT) of his head showed decreased attenuation in the right frontal parietal area, compatible with recent infarction. Intravenous digital subtraction angiography performed subsequently showed occlusions of both common carotid arteries. In May 1987, he began to have spells of bilateral loss of vision associated exclusively with exposure to bright light, particularly sunlight. Each episode persisted until he got into a darker environment; then it would take up to an hour for his vision to return completely to normal. The vision loss occurred as a general darkening in both visual fields over several seconds. He could avoid spells by using wraparound sunglasses or a baseball cap when going outside.

On examination at the Mayo Clinic on June 3, 1987, he had a right brachial blood pressure of 146/100 mm Hg and a left brachial pressure of 210/82 mm Hg. There was a grade 2/6 diffuse systolic bruit over the right carotid artery and a grade 1/6 systolic bruit over the proximal left subclavian artery. Ophthalmoscopy revealed bilateral venous-stasis retinopathy (Figure 1). Visual acuity was 14/21 bilaterally with correction.

Ocular pneumoplethysmography revealed very low values bilaterally (39 mm Hg on the left and 30 mm Hg on the right), consistent with bilateral carotid artery system occlusive disease.

Treatment was aspirin orally. Nine months later, the spells were unchanged in frequency or severity. He no longer was driving his car at night because exposure to bright car lights decreased his visual
acuity. He had no spells of focal cerebral ischemia while on the aspirin regimen.

Case 2

A 68-year-old man experienced episodes of bilateral blurring and dimming of vision beginning in 1983. These episodes occurred only when he was looking at bright light (approximately 90% occurred on exposure to sunlight) and persisted as long as he looked at the light; they resolved within 1-2 minutes after the exposure ended. A typical spell began with “black spots” throughout the visual field and a subacute generalized decrease in visual acuity and brightness progressing over approximately 30 seconds after the light exposure began. The episodes were less severe if he wore sunglasses.

Neurologic examination at the Mayo Clinic in June 1986 revealed bilateral localized systolic carotid bruits of grade 3–4/6 on the left and grade 2/6 on the right. Visual acuity was normal bilaterally, but ophthalmoscopy revealed bilateral mild venous engorgement and a soft exudate superior and temporal to the left disk. Intraocular tensions were 8 mm Hg on the right and 10 mm Hg on the left.

Ocular pneumoplethysmography revealed very low ophthalmic systolic pressures bilaterally (86 mm Hg). Transaxillary cerebral angiography (Figure 2) revealed high-grade stenosis at the origin of both internal carotid arteries and mild stenosis at the origin of the left external carotid artery. A small right vertebral artery ended predominantly in the posterior inferior cerebellar artery, and the left vertebral artery had an irregular atherosclerotic plaque causing approximately 40% stenosis in the distal portion.

He underwent left carotid endarterectomy in July 1986, without complications. Electroencephalographic monitoring indicated that collateral blood flow was adequate during the acute period of clamping of the left carotid artery. Blood flow before clamping, during clamping, and during shunting was 8, 30, and 17 ml/min/100 mg, respectively. Ocular pneumoplethysmography on postoperative Day 1 gave normal results on the left and abnormal results on the right (ophthalmic systolic pressures were 93 mm Hg on the right and 119 mm Hg on the left, with brachial systolic pressure of 174 mm Hg).

Over the 21 months since carotid endarterectomy, he has had no spells of vision loss with or without exposure to bright light and no episodes of focal cerebral ischemia.

Case 3

An 82-year-old man began experiencing episodes of bilateral blurring of vision exclusively with exposure to bright light in July 1985. The episodes occurred in more than half of the times he was exposed to bright sunlight and resolved within seconds after the exposure ended. There were no other neurologic symptoms.

Examination at the Mayo Clinic on July 15, 1986, revealed a grade 1/6 systolic low-pitched localized right carotid bruit. Blood pressure was 160/90 mm Hg
FIGURE 2. Case 2. Transaxillary cerebral angiograms. Right lateral (top left) and anteroposterior (top right) views after common carotid artery injection. These studies indicate 99% stenosis of right internal carotid artery with string sign extending from origin of right internal carotid artery to cavernous segment. Left lateral (bottom left) and anteroposterior (bottom right) views after left common carotid artery injection, showing 99% stenosis at origin of left internal carotid artery and mild stenosis at origin of left external carotid artery.

in both arms sitting and 100/70 mm Hg in the left arm standing. Ophthalmoscopy revealed dilated retinal veins, particularly on the left.

Ocular pneumoplethysmography gave results consistent with a significant lesion of the right internal carotid artery system (ophthalmic systolic pressure...
of 115 mm Hg on the right and 134 mm Hg on the left, brachial systolic pressure of 180 mm Hg). Transfemoral cerebral angiograms showed occlusion of the right internal carotid artery approximately 1.5 cm distal to its origin and approximately 75% ulcerated stenosis of the left common carotid artery and of the left internal carotid artery at the bifurcation. A 30% left carotid siphon stenosis also was identified.

On July 21, 1986, he underwent left carotid endarterectomy with intraoperative electroencephalographic monitoring, without complications. The electroencephalograms demonstrated that collateral blood flow to both hemispheres was inadequate during the first clamping of the left carotid artery, but normal symmetric patterns were promptly restored by the shunting used during the procedure.

Postoperatively, his spells were less frequent and less severe. Twenty months later, his spells continue to be less severe than preoperatively, but otherwise their character has remained unchanged. He has experienced no other spells of focal cerebral ischemia since the operation.

Case 4

An 81-year-old man began experiencing bilateral fogging in both eyes in January 1987. These episodes consisted of a general dimming of vision lasting 5–15 minutes and were exclusively related to exposure to bright sunlight; they occurred with most exposures to sunlight.

At examination on June 4, 1987, bilateral localized grade 2/6 systolic carotid bruits were detected. Ophthalmoscopy showed vessels of normal caliber without venous-stasis retinopathy. Visual acuity was 14/21 on the right and 14/56 on the left.

Ocular pneumoplethysmography revealed low ophthalmic arterial pressures bilaterally (81 mm Hg on the right and 73 mm Hg on the left, with brachial systolic pressures 136 mm Hg on the right and 142 mm Hg on the left). Previous studies in 1981, 1984, and 1985 had given normal results. Digital venous angiography revealed high-grade stenosis in the proximal internal carotid arteries on both sides, with diminished perfusion in both cerebral hemispheres on the intracranial view.

The patient was treated with aspirin (325 mg/day). Three months later, his visual symptoms were unchanged. He died suddenly, on November 1, 1987, of an acute myocardial infarction. No postmortem examination was performed. By history there had been no intervening episodes of focal cerebral ischemia.

Discussion

The most typical form of amaurosis fugax consists of sudden unilateral vision loss unrelated to exposure to bright light. These episodes have often been associated with ipsilateral atherosclerotic carotid stenosis, with or without ulceration, with retinal microembolization. Episodes usually consist of a loss of vision developing over seconds and lasting for several seconds or minutes. The patient may describe the experience as a curtain or shade obscuring part or all of the vision in one eye, or it may progress from peripheral to central in the affected visual field. The loss of vision may be total or partial, and dimming of vision may occur in the upper or lower half of the visual field. Less commonly, scintillations or other positive visual phenomena may occur.

The phenomenon of unilateral vision loss associated with exposure to bright light has been associated with carotid artery occlusive disease; however, this phenomenon has also been associated with macular disease such as chorioretinitis or retinal pigmentary degeneration.

Our cases show that bilateral loss or dimming of vision in response to exposure to bright light may occur with bilateral carotid occlusive disease. The symptoms are apparently related to bilateral simultaneous retinal ischemia from reduced choroidal (choriocapillaris) blood flow delaying regeneration of visual pigments in the pigment epithelial layer. This is particularly pertinent in patients with suspected cerebrovascular disease because transient episodes of simultaneous bilateral vision loss usually have been associated with bilateral occipital lobe ischemia from vertebrobasilar occlusive disease. All four of our patients were thought to have had posterior circulation ischemic events related to vertebrobasilar atherosclerotic disease or migraine at some point in their clinical course.

Three of the four patients had at least some evidence of venous-stasis retinopathy, another indicator of retinal ischemia. Nevertheless, other cases of unilateral vision loss with light exposure and ipsilateral carotid artery stenosis or occlusion have been documented without evidence of venous-stasis retinopathy. Conversely, most patients with venous-stasis retinopathy do not have the phenomenon of light-induced vision loss.

Two patients underwent carotid endarterectomy, and two were treated with aspirin. Both of the former experienced improvement: one had complete resolution of the episodes of vision loss, and the other had partial resolution. Both patients treated with aspirin had no change in their episodes, and one died of acute myocardial infarction 11 months after starting treatment. None of the four patients had episodes of focal cerebral ischemia after starting treatment, and none had retinal infarction.

Clinical features characterizing the bilateral vision loss in this group of patients included 1) episodes occurred exclusively with exposure to bright light; 2) episodes occurred with most or all exposures to sunlight; 3) onset of vision loss was blurring, dimming, or scotomata in both eyes (never a typical shade effect) over several seconds to minutes (generally longer than typical episodes of unilateral amaurosis fugax); 4) episodes persisted for as long as the patient was exposed to bright light and for seconds to hours after the exposure; and 5) epi-
sodes uniformly were diminished by the use of sunglasses.

These episodes can be distinguished clinically from typical amaurosis fugax by their bilaterality, their exclusive relationship to bright light, their lack of a typical shade effect, and their longer duration of onset of the loss. Episodes of vision loss as a consequence of bilateral occipital lobe ischemia from vertebrobasilar system disease usually are bilateral and, although they may begin with generalized blurriness, dimness, or scotomata, they often involve combinations of right or left hemianopsias or quadrantanopsias with vertical midline splits to field defects with or without sparing of central vision. The onset of this vision loss is usually more rapid and, most important, is not related to exposure to bright light. Patients with bilateral occipital lobe ischemia also often have other symptoms of vertebrobasilar system ischemia.

References

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