

Transient monocular obscuration—? amaurosis fugax: a case report

GREGORY S KOSMORSKY,¹ STEVEN I ROSENFELD,¹ AND RONALD M BURDE^{1,2}

From the ¹Departments of Ophthalmology and Neurology and ²Neurological Surgery, Washington University School of Medicine, St Louis, Missouri, USA

SUMMARY A 73-year-old white man with pseudophakia experienced repeated bouts of transient visual loss associated with erythropsia and colour desaturation. A diagnosis of atheromatous carotid vascular disease was considered, prompting carotid angiography, during which time the patient experienced transient aphasia. Subsequent examination during an episode of visual loss showed that a spontaneous anterior chamber haemorrhage was the cause of the visual complaints.

The complaint of transient bouts of monocular visual loss in an older person suggests the presence of atheromatous disease of the carotid arteries and the necessity for neuroradiological examination. The symptoms during an attack of transient monocular visual loss may suggest various diagnoses and obviate the need for such studies, with their inherent risk. A patient with complaints of recurrent bouts of transient monocular visual loss and suffering from temporary speech arrest followed by mild aphasia subsequent to carotid angiography is the subject of this report.

Case report

A 73-year-old white man began experiencing bouts of transient visual blurring affecting the right eye eight months before his initial examination. During these eight months repeated examinations by his ophthalmologist while the patient was asymptomatic failed to reveal any ocular pathology. He was referred to an internist and then to a neurologist for evaluation of 'amaurosis fugax.' Venous subtraction angiography revealed irregularities in the area of the left carotid bifurcation. Subsequent carotid angiography showed ulceration and mild stenosis of the left internal carotid artery at the bifurcation and a 50% stenosis of the carotid siphon on the left side. The right carotid

bifurcation was normal, there was only moderate narrowing of the siphon, and no ulceration was seen. During the carotid angiography, after repositioning the catheter in the aortic arch, the patient noted he was unable to talk for a short time, and several minutes of aphasia followed. After this episode a computerised axial tomogram was normal. The patient was treated with dipyridamole (Persantine) and aspirin without any alteration in his symptoms.

Elucidation of the patient's history revealed that the episode of so-called monocular visual loss consisted of a sensation of 'foggy vision' associated with erythropsia (seeing red) and colour desaturation, especially for the colour red. He specifically denied total blackouts or a curtain. These episodes initially lasted 15–20 minutes and occurred once a month, but by the time of his referral his longest episode had lasted seven hours and the frequency had increased to three to four bouts per week. The episodes were sudden in onset and unassociated with any neurological symptoms such as weakness, numbness, dysarthria, diplopia, headache, or orbital pain. No precipitating events could be identified. According to his medical history he had had an intracapsular cataract extraction with iris fixation lens implant in the left eye in 1973, and a similar procedure had been performed in the right eye in 1980, both without complications. Topical 2% pilocarpine had been used intermittently to stabilise the lens in his right eye. He also had a history of hypertension for 10 years, treated with chlorothiazide (Diuril), and arthritis occasionally treated with aspirin.

Correspondence to Ronald M Burde, MD, Department of Ophthalmology—Box 8096, 660 South Euclid Avenue, St Louis, MO 63110, USA.

Transient monocular obscuration

Examination at the time of referral showed a visual acuity of 20/20 in the right eye and 20/15 in the left eye at distance. His right pupil was 3 mm, square (from an iris fixation implant), and non-reactive. The left pupil was 4 mm and minimally reactive. The results of tests of optic nerve function, brightness and colour comparison, and American Optical colour plates were equal and normal bilaterally. Amsler grid testing revealed no defects. Slit lamp examination showed well healed anterior segments on both sides. The intraocular lens implants did not come close to any angle structures, and the foot processes were not associated with any iris or pupillary lesions. Applanation pressures were 18 mmHg in the right eye and 21 mmHg in the left. Visual field testing by kinetic perimetry was normal. Gonioscopy performed at this time revealed an abnormal sheath of vessels near the iridectomy in the superior angle of the right eye. Ophthalmodynamometry was 60 mmHg in both eyes.

The patient was subsequently seen again during an attack of blurred vision. Examination then revealed diffuse anterior chamber haze secondary to a considerable number of erythrocytes in the aqueous, which were also found to be layered on the corneal endothelium. In addition gonioscopy performed then showed blood streaming from the previously noted vessels in the superior chamber angle and a microscopic hyphaema in the inferior angle. The patient refused fluorescein angiography because of a possible allergy to contrast material.

Discussion

The classic features of amaurosis fugax include sudden painless visual loss in one eye which lasts from 30 seconds to 15 minutes (usually 2 to 3 minutes) and then vision returns.¹ The visual loss may be total or partial, often being described as a 'shade effect' marked by a sharp horizontal border involving the upper or lower hemifield. The implication of this type of monocular visual disturbance is that of ipsilateral carotid atherosclerotic disease with or without ulceration, producing either a cholesterol plaque or a platelet fibrin embolus which lodges in the central retinal artery or one of its main branches. Thus, when the clinician hears that a patient has had bouts of transient monocular visual loss, he often immediately begins an investigation of the carotid system, including angiography with all of its attendant risks.

Our patient described the sudden onset of painless, recurrent monocular visual disturbance which consisted of erythropsia, colour desaturation, and a fogginess of his vision. The duration of symptoms ranged from 15 minutes to seven hours. These symptoms are in sharp contrast to those of classic

amaurosis fugax. To our knowledge, the complaint of erythropsia has not been reported with amaurosis fugax, and it should suggest either intraocular haemorrhage or bleaching of the aphakic or pseudophakic retina after exposure to bright sunlight. In addition, most patients with occlusive carotid disease and amaurosis fugax experience other concomitant transient neurological symptoms, though a small percentage will have only visual complaints. Thus, the lack of associated neurological symptoms in our patient suggests, but is not diagnostic of, a normal carotid circulation. The history is thus the key to suspecting the diagnosis of an anterior chamber haemorrhage. Most often when the patient is asymptomatic the ophthalmological examination will be entirely normal. In fact the anterior chamber may clear within hours of a symptomatic haemorrhage.² Therefore the clinician needs a high index of suspicion of the possibility of a transient anterior chamber haemorrhage.

The causes of spontaneous anterior chamber haemorrhages are numerous (Table 1). Such haemorrhages may be seen as a late complication of cataract surgery due to ruptured incisional vessels,^{3,4} iris or angle neovascularisation, peripupillary microangiomas, coagulation disorders, or anticoagulation medication.⁵ In addition the implantation of an intraocular lens compounds the problem depending on the type of lens used. For example, the uveitis-glaucoma-hyphaema (UGH) syndrome^{6,7} may be produced by rigid anterior chamber lenses. Peri-

Table 1 Causes of spontaneous anterior chamber haemorrhages

Vascular anomalies of the iris
Myotonic dystrophy ^{11 12}
Sturge-Weber syndrome ¹³
Microhaemangiomas
Diffuse haemangiomas of childhood ¹⁴
Neoplasms
Melanoma
Retinoblastoma ¹³
Diseases of the blood or vessels
Leukaemia ¹³
Haemophilia ¹³
Scurvy ¹³
Lymphoma ¹⁵
Rubeosis iridis ¹⁶
Severe iritis ^{13 16}
Fibrovascular membranes
Juvenile xanthogranuloma ¹⁶
Occult trauma or delayed bleeding after trauma ¹⁶
Hydrophthalmos ¹⁷
Malignant exophthalmos ¹³
Histiocytosis X ¹⁸
Postsclerotomy with cautery ¹⁹
Postcataract surgery bridging wound vessels ³ ; irritative phenomenon with neovascular tufts associated with intraocular lenses ^{9 20}

pupillary iris erosion associated with either pupillary plane or posterior chamber lenses may produce similar clinical findings.⁸⁻¹⁰

In our case an abnormal tuft of vessels was noted near the iridectomy at the time of initial examination, and this proved to be the site of haemorrhage when the patient was examined during a bout of visual blurring. The cause of the neovascular tuft of vessels in this location is not clear. If the diagnosis of spontaneous intraocular haemorrhage had been made earlier, angiography with its associated risks might have been obviated. The clinician should be aware of the possibility of spontaneous anterior chamber haemorrhage as a cause of transient monocular visual disturbance. Erythroptosis should arouse the suspicion of intraocular haemorrhage. Ophthalmological examination, preferably while the patient has symptoms, may be diagnostic and obviate the need for invasive and potentially dangerous studies.

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