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Transient monocular blindness

The term amaurosis fugax, which means “fleeting blindness”, has come to be associated with transient monocular blindness due to embolism reaching the retinal circulation from the carotid vessels or from the heart. As amaurosis fugax may precede a stroke it is usually viewed in a neurological context. Transient monocular blindness, however, is not solely caused by emboli. It has many causes, including migraine, intracranial hypertension, and malignant hypertension and it is, therefore, important to make an accurate diagnosis. We describe a case of aophthalmic condition, intermittent angle closure glaucoma, giving rise to transient monocular blindness, so emphasising the need to be aware of such diagnoses.

A 66 year old woman presented with a 12 month history of repeated episodes of transient loss of vision in her right eye. The episodes were precipitated by reading, writing, and watching television for a variable time. They came on suddenly and she described a film descending over her right eye leading to complete loss of vision. The episodes lasted from three minutes to several hours. On one occasion she was an ambulance aache over the right side of the forehead. She had no positive visual symptomatology during an attack and there was no family history of migraine. She smoked six cigarettes a day. She had a history of palpitation and blackouts for which she attended a cardiologist. An ECG and an exercise stress test were normal; however, a 24 hour Holter monitor had disclosed frequent ventricular extrasystoles and an episode of atrial fibrillation. She was treated with a β blocker and subsequently amiodarone, but this was discontinued by the patient. Neurological examination was normal, as were carotid duplex scans, brain CT, and an echocardiogram. A provisional diagnosis of amaurosis fugax was made and she was started on 75 mg aspirin a day. In view of the specific predisposition for her condition, which was not clear, she was referred for a neuro-opthalmic opinion.

Initial neuro-opthalmic assessment was normal. After a diagnosis was made it was decided to try to precipitate an attack. After reading intermittently over a period of four hours she reported loss of vision in her right eye. Slit lamp examination showed pronounced corneal oedema on the right, a poorly reacting semidilated pupil and a shallow anterior chamber. Her intraocular pressures were 50 mm Hg on the right and 18 mm Hg on the left. Pulsation of the central retinal arteries was noted in both eyes.

A diagnosis of intermittent angle closure glaucoma was made. After initial medical treatment to constrict the pupil and lower the intraocular pressure, n-DY-LAG laser iridotomies were performed. Subsequent gonioscopy confirmed a narrow drainage angle and refraction disclosed a moderate degree of hypermetropia which may be associated with a shallow anterior chamber and narrow drainage angle. A new appointment three months later, the patient reported that since the iridotomies she had had no further episodes of visual disturbance.

The first report that transient monocular blindness could precede central retinal hemic- plegia was by Miller Fisher in 1952.1 He stated that “Blindness is usually complete in the affected eye, although at times the defect is so slight that only a momentary obliteration of vision of attacks varies from several a day to a few each year. Symptoms last for years or may disappear completely after a few months...The blindness most commonly comes on as an eye being being lowered or raised, and vision returns from the opposite direction...The attacks last from a minute or so up to seven minutes or more.”

This led to the awareness that transient monocular blindness may warn of an impending stroke and the need to institute preventative measures.

Our patient had many of the features reported by Miller Fisher. She also had several carotid arteriosclerotic risk factors, being a smoker and having a history of cardiac arrhythmias. She had features which suggested an alternative diagnosis, however. In particular the specific precipitating factors were atypical. The duration of attacks, which on occasions lasted several hours, was unusual, although attacks of amaurosis fugax of up to 24 hours have been reported.2 A diagnosis of angle closure glaucoma was not originally suspected, as on direct questioning the patient neither reported seeing haloes, nor having any visual loss associated with poor lighting conditions. Angle closure glaucoma may be associated with severe pain and injection of the globe, whereas our patient had complained only of an aching sensation and had not noted any redness of the eye.

Angle closure glaucoma has previously been reported as a cause of transient monocular visual loss.3 In the three cases reported the initial presenting diagnoses were either of amaurosis fugax or migrainous phenomen- ena. Two of the patients described seeing haloes, however, and none of them had specific precipitating factors such as reading. Close ocular work such as reading and sewing can precipitate angle closure glau- coma. It is, however, uncommon for reading to be the sole precipitating factor.

Angle closure glaucoma can cause visual loss by various different mechanisms. The raised intraocular pressure leads to corneal clouding due to oedema and may reduce the perfusion pressure of the eye, thereby impairing blood flow to the choroid, retina, and optic disc. A blind spot may be seen.

In conclusion, although emboli are responsible for most cases of transient monocular blindness other causes should always be considered (see review by GAUSTAD4), especially in the presence of atypi- cal features such as specific precipitating fac- tors or an unusually long duration of symptoms.

Acquired bilateral opercular lesions or Foix-Chavy-Marie syndrome and eating epilepsy

The Foix-Chavy-Marie syndrome or bilateral anterior opercular syndrome (AOS) consists of lower facial and glossopharyngeal diplegia secondary to dysfunction of the rolandic operculum. It is usually seen in adults but it can result from any condition but is mainly due to successive strokes involving both opercular regions.1 Children developing AOS acutely as a consequence of an acute CNS illness have been described.2 A 20 year old man had normal development until, at the age of 4 years, he developed acute, presumably viral, meningo-encephalitis. This led to a prolonged stay in hospital in an intensive care unit. He was left with severe dysphasia and bilateral tabsiobasalifosaglypharyngeal paresis. At the age of 11 he developed partial motor seizures (involving the left facial muscles) with occasional secondary generalisation. Attacks occurred from time to time and there was no relation with any specific foods, definite times, or other specific triggering factors. He had received different antiepileptic medications with poor results until com- bined treatment with carbamazepine (600 mg/day), valproate (1500 mg/day), and clobazam (15 mg/day) was initiated. He now has only one or two seizures a year, always triggered by eating.

On neurological examination his mouth was always open and he drooled continuously. He had bilateral lip, tongue, and pha-ryngeal weakness with dissociation of automatic and voluntary movements of the lower face (voluntary movements impaired and automatic movements preserved). Eye closure and extraocular movements were normal. Affect was normal. He had a brisk jaw jerk. Language was limited to guttural vowel sounds, but his comprehension was normal. He had a minimal left upper limb paresis and generalised hyperreflexia, more pronounced on the left. Computed tomogra- phy and MRI showed atrophic lesions involving both rolandic opercula (figure). Recordings from EEG showed normal back-
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