Transient monocular blindness

The term amaurosis fugax, which means "fleeting blindness", has come to be associated with transient monocular blindness due to emboli 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 a typical amnestic 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. She was an association of a ache 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 no personal or paternal history of palpebral myopathies or for which she attended a cardiologist. An ECG and an exercise stress test were normal; however, a 24 hour Holter monitor showed 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 presence of her intracranial transient anemia she was referred for a neuro-ophthalmic opinion.

Initial neuro-ophthalmic assessment was normal. A diagnosis of a 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. Sit 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 artery was normal 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, Nd:YAG 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. Four week 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 contralateral hemi-plegia was by Miller Fisher in 1952. He stated that "Blindness is usually complete in the affected eye, although at times the defect is fluctuating. The frequency 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 during the day, the eye 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 cardiovascular 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. 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 is 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. In the three cases reported the initial presenting diagnoses were either of amaurosis fugax or migrainous phenomena. 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 glaucoma. 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 IOP leads to blind spots, a clouding due to oedema and may reduce the perfusion pressure of the eye, thereby impairing blood flow to the choroid, retina, and optic disc (Gruessner & Koller, 1982; Sabti & Aigner, 1982).

In conclusion, although emboli are responsible for most cases of transient monocular blindness other causes should always be considered (see review by Gass, 1991), especially in the presence of atypical features such as specific precipitating factors or an unusually long duration of symptoms.

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

The Foix-Chavany-Marie syndrome or bilateral anterior opercular syndrome (AOS) consists of lower facial and glossoparesial diplegia secondary to dysfunction of the rolandic operculum. It is usually seen in adults. It can result from focal or diffuse lesions and is mainly due to successive strokes involving both opercular regions. Children developing AOS acutely as a consequence of an acute CNS illness have been described. A 20 year old man had normal development until, at the age of 4 years, he developed acute, presumably viral, meningencephalitis. This led to a prolonged stay in hospital in an intensive care unit. He was left with severe dysphasia, bilateral focal right hemisphere

Correspondence to: Dr Kwai Fu Ko, Block 5, Flat D, 19/F, Garden 23 Electric Road, North Point, Hong Kong.


EDIN O'SULLIVAN
SANDIP SHAUNAK
TIMOTHY MATTHEWS
JOHN WADE
CHRISTOPHER KENNARD
Academic Unit of Neuroscience, Charing Cross and Westminster Medical School, Fulham Palace Rd, London W6 8RF, UK.


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

The Foix-Chavany-Marie syndrome or bilateral anterior opercular syndrome (AOS) consists of lower facial and glossoparesial diplegia secondary to dysfunction of the rolandic operculum. It is usually seen in adults. It can result from focal or diffuse lesions and is mainly due to successive strokes involving both opercular regions. Children developing AOS acutely as a consequence of an acute CNS illness have been described. A 20 year old man had normal development until, at the age of 4 years, he developed acute, presumably viral, meningencephalitis. This led to a prolonged stay in hospital in an intensive care unit. He was left with severe dysphasia, bilateral focal right hemisphere
Transient monocular blindness.

E O'Sullivan, S Shaunak, T Matthews, J Wade, C Kennard and P Simcock

*J Neurol Neurosurg Psychiatry* 1995 59: 559
doi: 10.1136/jnnp.59.5.559

Updated information and services can be found at:
http://jnnp.bmj.com/content/59/5/559.1.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/