Sudden onset double vision

Eye series – 11

Chris Hodge, BAppSc, DOBA, is Research Director, The Eye Institute, Chatswood, New South Wales.
Frank Martin, MBBS, FRANZCO, FRACS, is an ophthalmic surgeon, Sydney, and The Eye Institute, Chatswood, New South Wales.

A 50 year old woman presents complaining of double vision. She had become aware of double images while watching television the previous night. There was no associated pain or headache. Apart from being overweight the patient is relatively healthy. She is on no current medication. There is a strong family history of diabetes. On observation the patient’s right eye is turning inward (Figure 1).

Question 1
What are the main causes of acquired double vision?

Answer 1
Acquired double vision can be either monocular or binocular in nature. Monocular diplopia is commonly due to abnormalities of the eye:
- uncorrected refractive error (particularly high astigmatism)
- subluxated natural lens (systemic conditions such as Marfan’s syndrome)
- cataract
- retinal disease.
In the normal situation both eyes work together to form a single image. Binocular diplopia occurs when the relationship is disrupted. The breakdown may be either neurological or mechanical in origin. Causes include:
- cranial nerve palsies (from trauma, intracranial lesions, diabetes)
- extraocular muscle weakness (from cranial nerve palsy) exacerbates diplopia
- direct trauma to the eye (orbital wall fractures causing mechanical restriction or entrapment of the extraocular muscles).
It is important to differentiate between diplopia and a distortion of the vision (metamorphopsia) as commonly produced by retinal disorders such as age related macular degeneration. This should become apparent on further questioning.

Answer 2
Careful history taking will help define the patient examination and may be as important as the clinical tests. The following questions will help provide direction to the final diagnosis:
- monocular or binocular – if the diplopia continues when one eye is closed the condition is monocular. If the diplopia disappears it is due to a breakdown in the binocularity of both eyes
- onset – sudden onset may indicate vascular origin while gradual deterioration may be due to a progressive neurological disorder such as progressive supranuclear palsy or a slow growing tumour
- vertical or horizontal – the direction of the double image may indicate the specific involvement of an extraocular muscle. (Similarly some extraocular muscles will dominate with either close or distance vision. For example, a sixth nerve or lateral rectus palsy will be more noticeable when the patient is viewing a...
Sudden onset double vision

Reprinted from Australian Family Physician Vol. 31, No. 12, December 2002 • 1017

Neurological disorders usually lead to vertical diplopia
• associated symptoms – motor symptoms such as ataxic gait may indicate an associated progressive neurological disorder.

Answer 3

The extraocular muscles are controlled by three cranial nerves:
• the oculomotor nerve (third) controls the superior, inferior and medial recti muscles as well as the inferior oblique muscles. A complete third nerve palsy will leave the patient with the affected eye turning down and outward. (The patient will also have a lid ptosis and dilated pupil)
• the trochlear or fourth nerve controls the superior oblique muscle. A patient with a fourth nerve palsy will often present with a hypertropia of the affected eye (one eye higher than the other in primary or normal gaze). To compensate for the loss of movement the patient will commonly tilt their head to the opposite shoulder
• the abducens, or sixth nerve, controls the lateral rectus. A sixth nerve palsy will cause the eye to turn inward, increasing when looking to the affected side.

Answer 4

The most common cause of sixth nerve palsy is microvascular disease. The patient may present with a history of diabetes, hypertension or hypercholesterolaemia. Giant cell arteritis can also lead to an acquired palsy. Tumours, viral disease (eg. meningitis), elevated intracranial pressure and various inflammatory disorders (eg. multiple sclerosis) are also known causes of acquired abducens palsy. Although more common in fourth cranial nerve palsies trauma may also lead to an acquired sixth palsy.

It is important to differentiate between true sixth nerve palsy and conditions that may also lead to decreased horizontal movement. Orbital wall fractures or intraorbital growths may inhibit lateral rectus movement and mimic abducens palsy (Figure 2). Myasthenia gravis can also produce similar symptoms although presentation is variable with periods of exacerbations and remissions. This would help to serve as a differential diagnosis.

Answer 5

If the underlying cause remains undetermined further investigation is required. A full screening for diabetes and hypertension should be undertaken. If the patient is over 60 years of age an erythrocyte sedimentation rate (ESR) test is indicated to rule out giant cell arteritis. If results return negative more descriptive tests such as CT or MRI scans should be completed to rule out potential neurological lesions. The presence of additional neurological signs should represent an emergency and require urgent consultation with a neurologist.

Answer 6

An acquired sixth nerve palsy of microvascular origin will usually resolve in 3–6 months without treatment. If the underlying cause is due to a lesion, the defect will not resolve until this is removed. About one-third of patients may experience a further episode at a later stage. Similarly a palsy of viral origin retains an excellent prognosis for recovery.

Answer 7

The patient should be followed regularly during the initial stages of the condition to monitor change and potential recovery. To help correct the diplopia, stick-on prisms (Fresnel prisms) can be applied to glasses to realign the images and provide comfort to the patient. These prisms will need to be adjusted regularly as the eye recovers. If the patient finds the prisms difficult to use or confusing, occlusion of the affected eye will serve to remove the diplopia albeit at the loss of depth perception. These patients should be warned about the potential hazards of operating with only one eye (eg. difficulty negotiating stairs or judging distances when driving). Botulinum toxin may be used in special cases to reduce the possible contracture of the opposing muscle (medial rectus). Contracture of the muscle may reduce the effectiveness of future surgery.

Because the majority of acquired palsies recover naturally, surgery should not be considered unless the condition has stabilised or muscle function assessed as permanently reduced at 12 months. The aim of surgery is to restore binocular vision in the primary position and assist further muscle movement thereby increasing the area of single vision and improving patient comfort. This should be the final option.

Conflict of interest: none declared.

Figure 2. MRI of orbital lesion leading to disruption of ocular movement through lateral rectus involvement