Diplopia: Diagnosis and management

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ABSTRACT

Diplopia or double vision is the separation of images vertically, horizontally or obliquely and can be monocular or binocular in origin. Binocular diplopia is most commonly caused by ocular misalignment or strabismus that can be detected using simple clinical tests. All patients with diplopia of acute onset should be investigated urgently and those with a headache or pupillary involvement need to be referred for same-day urgent imaging. Diplopia secondary to microvascular causes on the other hand often spontaneously resolves within six months.

Introduction

Diplopia or double vision is a condition in which an object is seen as a double image. The two images can be of the same intensity, or one may appear as a ghost or faint image. They can be seen side-by-side, one on top of the other or tilted, and this can vary in different directions of gaze. Diplopia is usually binocular when it occurs only when both eyes are open and disappears when one is closed, or monocular where it persists even after closing one eye. It may be a benign condition but at times may herald a significant underlying condition that requires investigation and treatment.[1]

The physiology of binocular vision

Binocular vision is a complex phenomenon that relies on several components working together. The eyes are moved by six extra-ocular muscles: the lateral rectus (LR), medial rectus (MR), inferior rectus (IR), superior rectus (SR), inferior oblique (IO) and superior oblique (SO). These in turn are controlled by three cranial nerves, termed the third, fourth and sixth cranial nerves, with the sixth nerve supplying the LR, the fourth the SO and the third the MR, IR, SR and IO. These cranial nerves arise in the midbrain (third and fourth nerves) and pons (the sixth nerve), with the nuclei linked by the medial longitudinal fasciculus.

The output from these in turn is coordinated by the supranuclear gaze centres. The horizontal gaze centre is in the pons in the parapontine reticular formation (PPRF) and the vertical gaze centre is in the midbrain reticular formation and the pretectal area. In addition, images are affected by head position, governed by the oculovestibular reflex (VOR). By adjusting the position of the eyes to counteract the movement of the head, the VOR stabilises the image on the retina and prevents blurring. These images are relayed to the brain through the optic nerve and the visual pathways, where visual processing occurs to form a single composite image.

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Causes of diplopia

Binocular diplopia

In normal viewing conditions, each eye sends a slightly different image to the brain and the cortical fusion mechanisms join the two images together, utilising the slight disparity between them to create the illusion of 3-D vision or stereopsis. If the images sent to the brain are too dissimilar due to misalignment of the eyes, diplopia results. This misalignment of the eyes is called strabismus (squint) and may be vertical, horizontal or torsional. Strabismus may occur secondary to neurological or mechanical factors and is almost always acquired if associated with diplopia.

Rarely, following trauma or central neurological insult such as a cerebral vascular accident, the brain loses its ability to fuse these images despite good alignment (horror fusionis).[2]

Monocular diplopia

This variety of double vision persists even if one eye is closed and is associated with ocular changes such as dry eyes, corneal scarring, cataract, retinal membranes or non-organic causes.[3]

Key points

- Diplopia may be binocular or monocular and can be differentiated by asking the patient to close one eye.
- Diplopia can be vertical, horizontal, torsional or a combination of these.
- It is helpful to evaluate the ocular deviation and motility in all cases of diplopia to determine the aetiology.
- Acute onset of binocular diplopia is a red flag and needs to be investigated.
- A dilated pupil or severe headache associated with diplopia is an emergency and needs urgent imaging.

KEYWORDS: strabismus, diplopia, binocular

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History taking

It is essential to cultivate a logical and structured approach to history taking and examination in patients with diplopia. While each case is distinctive, using a logical approach will ensure that important clinical information is not missed and an accurate diagnosis can be reached. Diplopia, especially of acute onset, is a red flag and the possibility of an underlying neurological cause should be strongly considered.

Presenting complaints

Is the onset of double vision recent (which may indicate a neurological event) or long-standing (decompensated strabismus, which is less urgent)? Patients may recall an antecedent event such as head trauma, cerebrovascular event or sinus surgery.

Does the double vision disappear after closing one eye (binocular diplopia, which is more likely to require intervention) or persist (monocular diplopia, which is likely to be due to local ophthalmic or non-organic causes)?

Are the images separated horizontally (suggesting decompensated divergent or convergent squint, sixth nerve palsy, or internuclear ophthalmoplegia secondary to multiple sclerosis) or vertically (suggesting third and fourth nerve palsy or restrictive disorders such as blow out fracture or thyroid eye disease)?

Are there any associated symptoms such as ptosis (third nerve palsy, myasthenia), blurred vision (third nerve palsy due to dilated pupil), headache (raised intracranial pressure), weakness, fatigue, or difficulty in swallowing (myasthenia, demyelinating disorders), oscillopsia (due to nystagmus in internuclear ophthalmoplegia), lid retraction or proptosis (thyroid eye disease), temporal headache and pain while swallowing (temporal arteritis or giant call arteritis [GCA]) or abnormal head posture?

Previous ophthalmic history

Ask specifically about the use of glasses/contact lenses and whether their use makes the strabismus better (which suggests decompensated longstanding strabismus). Are they using any prisms in their glasses? Do they have a history of occlusion of either eye in childhood signifying underlying amblyopia (which suggests longstanding strabismus)?

Have they experienced any previous trauma to the eye, or undergone any refractive or strabismus surgery?

Medical history

It is important to identify disorders such as hypertension and diabetes, previous cerebrovascular accidents and vascular risk factors. These are especially important to ascertain causation in cranial nerve palsies, as most vascular nerve palsies recover in the first 6 months and therefore only require observation or noninvasive treatment. Other systemic conditions associated with diplopia include hyperthyroidism and myasthenia gravis.

Of note, headache and pupil involvement may be seen in microvascular nerve palsies as well as surgical cranial nerve palsies and thus this does not help to distinguish the two.

Drugs such as lamotrigine, topiramate, gabapentin, fluroquinolones, and citalopram have been associated with diplopia, but this is a rare occurrence.

Family history

A family history of strabismus, amblyopia and high refractive errors may signify a genetic component.

Examination

Assess visual acuity with distance glasses or contact lenses in each eye. Close each eye to investigate if the diplopia is monocular or binocular.

Carry out a corneal reflex test by shining a torchlight from about 33 cm so that it can be seen reflected in both pupillary areas. This will immediately detect any obvious strabismus and estimate the size of deviation (30 dioptres if reflex is at edge of pupil, 45 dioptres if between pupil and limbus and 60 dioptres if at edge of limbus).

In a sixth nerve palsy the eye will be deviated inwards, in a third nerve palsy it will be deviated in a down and out position, and in a fourth nerve palsy, the affected eye will be higher, especially in medial gaze.

Assess ocular motility by asking the patient to follow torchlight or fixation target (finger, pen) as you move it into all directions of gaze in the nine directions of gaze, in a cross-like pattern, asking them to say if the object goes double at any stage. If the patient describes diplopia, ask them if it is vertical or horizontal ('Are the images side by side or one on top of the other?') which will help elucidate the type of deviation (sixth nerve palsy leads to horizontal separation; fourth and third nerve palsy can cause both horizontal and vertical separation; thyroid eye disease and blow out fracture are predominantly associated vertical separation). Oblique diplopia signifies a vertical as well as a horizontal component. The separation will increase in the field of maximum limitation.

Ocular examination can yield useful clues to the associated pathology: ptosis suggests myasthenia or third nerve palsy, lid retraction suggests thyroid eye disease, nystagmus suggests demyelination, proptosis suggests thyroid eye disease, and papilloedema may indicate raised intracranial pressure.

A fixed dilated pupil associated with headache and diplopia is a neurosurgical emergency and necessitates urgent imaging.

Systemic examination

All patients with diplopia should undergo a full cranial nerve and peripheral nervous system examination, as multiple cranial nerve palsies may signify intracranial- or meningeal-based tumours, meningitis, polyneuropathy, multiple sclerosis or cavernous sinus lesion.[4,5] It is also useful to look for signs of thyroid dysfunction if indicated.

Investigations

It is helpful to measure blood pressure and blood glucose and carry out an urinalysis for suspected microvascular cases, thyroid function tests, or single fibre EMG of the orbicularis and anti-acetylcholine receptor antibodies for myasthenia.

Imaging (an MRI or CT angiography scan) may be indicated if the onset is acute and/or associated with neurological signs or papilloedema.

Management

All patients with new-onset diplopia should be advised to stop driving.[6] Most microvascular causes of diplopia can be

observed if the rest of the examination is normal as they usually spontaneously resolve within 6 months. They may benefit from referral to the ophthalmology department as the diplopia can be alleviated with prisms, patches or toxin in the meantime.

Urgent, same-day imaging should be sought for patients with a fixed dilated pupil, headache and diplopia. An acute medical or rheumatology referral should be made if there is suspicion of associated GCA. Other symptoms of GCA include loss of weight, night sweats, temporal headache, jaw claudication and visual loss. ■

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