Diplopia – a symptom to take seriously

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Diplopia (double vision) can be the first, or the only, symptom of life-threatening disease such as expanding intracranial aneurysm, brain tumour, myasthenia gravis or giant cell arteritis. All patients with new-onset diplopia should, therefore, be urgently assessed and investigated.

Case presentation

A 23-year-old woman presented to her GP with a two-day history of painless horizontal double vision. She first noticed this problem while watching television, when she saw two television screens. The GP found her visual acuity to be 6/6 in each eye and the eyes looked well aligned. The pupils were the same size in each eye, there was no ptosis and eye movements appeared to be normal.

The GP referred the patient to an optometrist, who checked the patient's eyes thoroughly and reported that no ocular disease was present. However, the optometrist suggested the GP perform a neurological assessment.

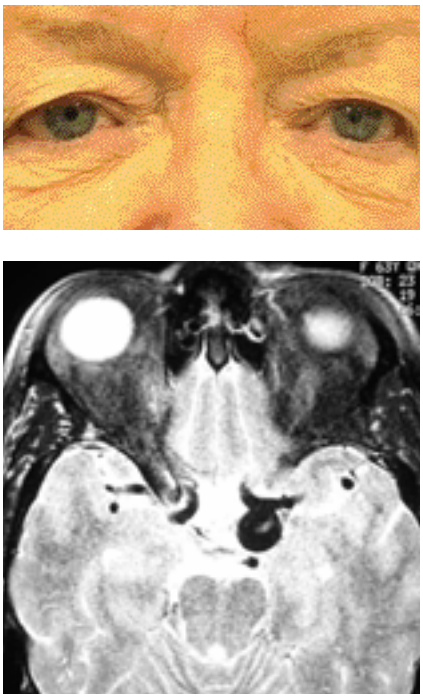
The patient's blood pressure was normal, she denied having any headaches, her gait was normal, and her limb power, sensation and deep tendon reflexes were also normal. The patient had been under considerable social stress recently and the GP thought her symptoms could be anxiety related. The patient was counselled to try and reduce her stress levels and it was suggested that she return to the GP in a week if the double vision persisted.

The patient returned the next day complaining of a worsening of the double vision and also pain behind the right eye. Examination was unchanged. The GP obtained a CT brain scan, which was normal. The patient was reassured but was routinely booked to see an ophthalmologist for further assessment.

That night the patient suddenly collapsed at home and was brought unconscious to the emergency department. MRI diagnosed a subarachnoid haemorrhage secondary to a large ruptured right posterior communicating artery aneurysm. Urgent neurosurgical intervention was attempted; however, the patient did not survive.

Discussion

This patient's diplopia was the result of an expanding aneurysm causing right partial third nerve palsy. Eye movement abnormalities resulting in double vision can be extremely subtle, and the eyes and eye movements often look normal on casual observation (Figures 1a to b). Some early partial third nerve palsies due to compression from an aneurysm or tumour are painless and have no associated drooping of the upper eyelid (ptosis). Contrary to popular teaching, the presence of normal-sized pupils (pupil sparing) in each eye does not exclude the possibility of an aneurysm or tumour.



Figures 1a to b. This 63-year-old woman presented complaining of slowly worsening vertical double vision for the past six months. a (top). Her eyes looked normal on casual examination: visual acuity was 6/6 in each eye; there was no ptosis; pupils were equal and reactive; the eyes looked normally aligned; and eye movements appeared full. b (bottom). Magnetic resonance imaging/angiography of the brain, however, revealed a large left posterior communicating artery aneurysm as the cause of the diplopia (by causing a subtle left partial third nerve palsy).

In cases of diplopia, it is important:

- not to suspect that the double vision is just an eye problem (and therefore unlikely to be serious)

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Table 1. Common causes of diplopia

- Posterior communicating artery aneurysm
- Brain tumour (benign or malignant)
- Myasthenia gravis
- Giant cell arteritis
- Stroke
- Ischaemic nerve palsies from atherosclerosis
- Thyroid orbitopathy
- Orbital fractures
- Congenital or traumatic fourth nerve palsy
- Decompensated phorias

Why is the cause of diplopia difficult to diagnose?

- Patients with serious causes of diplopia (e.g. aneurysm, tumour) may have eyes that 'look normal' on casual observation and 'follow my pen' eye movement testing.
- A thorough assessment of a patient with double vision is a complex process that requires specialised equipment, can take more than an hour, and is challenging even for an experienced ophthalmologist.
- Many different diseases can cause double vision and many can look the same on a superficial examination – there are often no 'spot diagnoses'.
- Warning symptoms and signs (e.g. headache, limb weakness and 'blown' pupil) are usually absent, and a patient with diplopia due to an aneurysm or tumour may otherwise seem well.
- Time may be short – a patient presenting with diplopia due to an expanding aneurysm may only have hours to live if it is left undiagnosed and untreated.

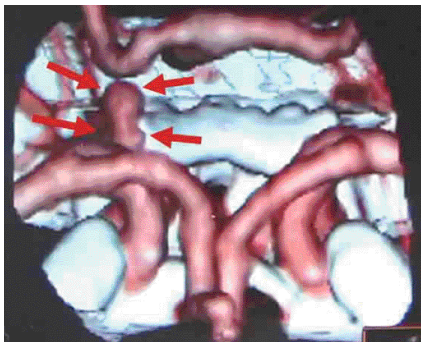
- not to think that if there is a serious cause for the double vision, other neurological symptoms or signs would be present (this is often not the case – in the patient described, the only structure compressed by the expanding aneurysm prior to its rupture was the right third nerve)
- not to presume that a CT brain scan will detect any serious pathology (CT scans alone will miss most aneurysms and also many brain tumours)
- to refer the patient with adequate urgency for evaluation by the appropriate specialist.

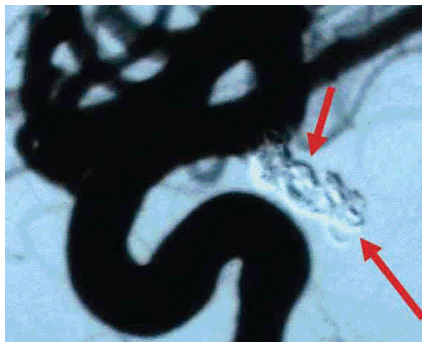
What diseases can cause diplopia?

Double vision due to misaligned eyes (binocular diplopia) can be due to the presence of disease anywhere in the eye movement chain of command – that is, the brain, ocular motor nerves (III, IV, VI), neuromuscular junction, extraocular muscles or orbits (Table 1). A final less common cause of double vision is monocular diplopia, in which a double image is seen just from one eye, for example because of a cataract.

Common causes of binocular diplopia include:

- posterior communicating artery aneurysm – this causes one-third of all third nerve palsies (Figures 2a to c). Patients may present with the following: vertical, horizontal or oblique diplopia; with or without ptosis; normal or dilated pupils; with or without headache or other neurological signs. Diplopia may have a sudden onset (with a rapidly expanding or rupturing aneurysm) or an insidious onset with slow progression
- brain tumours – most often sphenoid wing (Figures 3a to d) or suprasellar meningiomas, pituitary tumours or, less commonly, primary or metastatic malignancies. Diplopia usually has a gradual onset with a slow progression – except in pituitary apoplexy (sudden haemorrhagic infarction of a pituitary adenoma that presents with sudden headache, diplopia and loss of vision). Tumours



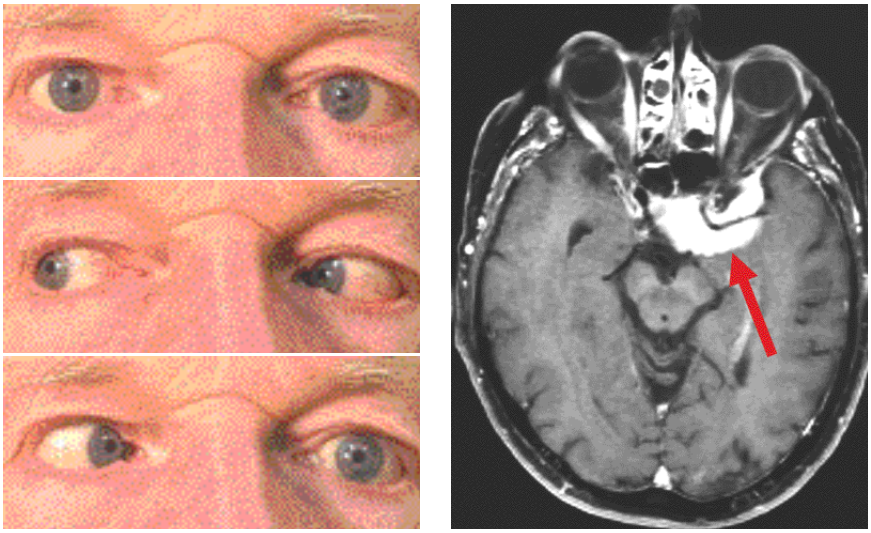


Figures 2a to c. This 52-year-old woman presented with sudden onset vertical diplopia. a (left). Examination revealed reduced elevation of the right eye and also a dilated right pupil. b (middle). CT angiography revealed a large right posterior communicating artery aneurysm (arrows). c (right). Insertion of platinum coils into the aneurysm via a femoral artery catheter approach (arrows) closed the aneurysm and saved the patient's life.

Table 2. Summary of the management of diplopia

- Urgent referral of the patient to a specialist
- Investigations may include:
  - MRI/MRA of the orbits and brain with contrast
  - blood tests including thyroid function tests and measurement of the levels of acetylcholine receptor antibodies, CRP and ESR
- Treatment may include:
  - treatment of the cause – e.g. neurosurgery, myasthenia gravis treatment
  - treatment of residual diplopia – e.g. prisms in spectacles, eye muscle surgery

ABBREVIATIONS: CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; MRA = magnetic resonance argiography; MRI = magnetic resonance imaging.



Figures 3a to d. This man noticed slowly worsening horizontal double vision, worse on looking to the left. a (top), b (middle), c (bottom). Examination showed a left sixth nerve palsy with reduced abduction of the left eye on leftwards gaze. d (above right). MRI revealed a large sphenoid wing meningioma (arrow).

can cause third, fourth or sixth nerve palsies or a combination of these.

- myasthenia gravis – ocular (confined to the eye muscles only) or generalised. Double vision is the presenting symptom, or the only symptom, in 50% of patients who have this disease. Diplopia can be horizontal, vertical or oblique; the patient may or may not also have a unilateral or bilateral ptosis.

Less common but equally dangerous causes of binocular diplopia include:

- giant cell arteritis – can cause transient or persisting double vision of any type in patients over 50 years of age
- brainstem stroke
- meningitis of any cause – can cause unilateral or bilateral third, fourth or sixth nerve palsies.

Common, relatively benign causes of binocular diplopia include:

- ischaemic third, fourth and sixth nerve palsies due to hypertension, hypercholesterolaemia, diabetes, etc. However, a nerve palsy is not ischaemic just because the patient has vascular disease (patients with diabetes also get brain tumours and aneurysm)
- thyroid orbitopathy
- congenital fourth nerve palsy
- traumatic fourth nerve palsy
- decompensated phoria.

The reasons why the cause of diplopia is difficult to diagnose is outlined in the box on page 75.

Suggested clinical approach

A suggested clinical approach for patients presenting with diplopia is outlined below.

- Take double vision seriously – in almost all cases it is not an eye problem
- Ask about, and look for, other features of brain disease or myasthenia gravis; however, these are often absent
- If a patient has acute-onset diplopia, refer urgently because it is a potential neurosurgical emergency
- If a patient has gradual-onset diplopia, refer urgently to a specialist if the patient has unequal pupils, ptosis or neurological symptoms; otherwise refer promptly (ideally to be seen within a week).

The management of diplopia is summarised in Table 2.

Conclusion

Acute-onset diplopia should be considered to be due to an expanding intracranial aneurysm until proven otherwise, no matter what is found on examination. Gradual-onset, slowly progressive diplopia is often due to a brain tumour. All patients complaining of double vision should be evaluated promptly by an ophthalmologist and, if other neurological symptoms are present, a neurologist as well. MT

Further reading

Pane AR, Burdon MA, Miller NR. The neuro-ophthalmology survival guide. Edinburgh: Mosby; 2007.

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COMPETING INTERESTS: None.