Background
Diplopia can often be a diagnostic challenge in the general practice setting. Differentials broadly include orbital pathology and neuromuscular aetiologies. Familiarity with the causes and subsequent investigations can help deliver efficient and effective patient care.

Objective
This article presents a unique case to illustrate the diagnostic approach to diplopia and highlights a commonly encountered yet often overlooked condition as a differential to diplopia in general practice.

Discussion
John Murtagh’s manual identifies seven ‘masquerades’ as diagnoses that are commonly missed in general practice. The Murtagh model can be appropriately applied to diplopia in reminding practitioners of differentials that can easily slip from one’s mind, yet can be diagnosed promptly with simple investigations. Atypical presentations of common disease processes should always be considered within the diagnostic framework for practitioners.

Keywords
thyroid diseases; endocrine system diseases; vision disorders; eye diseases

Case study
An Aboriginal and Torres Strait Islander woman aged 45 years presented to a rural general practice with a 1-month history of diplopia. She complained of having double vision, which was relieved on closure of one eye. Pain, other visual disturbance, neurological and constitutional symptoms were absent and there was no preceding trauma. Her past surgical history included cervical cancer that was diagnosed and treated 2 years earlier, as well as a motor vehicle accident requiring maxillary plates at the age of 4 years. She had no past history of neurological conditions, diabetes or hypertension. She smoked 15–20 cigarettes per day and was a non-drinker.

On examination, horizontal nystagmus was noted in the left eye and ocular movements of the eye revealed a rightward deviation on right gaze, indicating possible pathology relating to the rectus muscles. No proptosis was evident. Pupils were equal and reactive. Visual field testing was normal. All other cranial and peripheral nerve examinations were unremarkable. She was otherwise well.

Question 1
What is the differential diagnosis?

Question 2
What investigations should be ordered?

Case study continued
The patient’s thyroid function test revealed hyperthyroidism:

- Decreased TSH <0.01 mU/L (0.27–4.20)
- Elevated free T4 at 23 pmol/L (12–22)
- Elevated free T3 at 7.04 pmol/L (4.0–6.8).

Computed tomography (CT) scan of the orbit shown in Figure 1 revealed asymmetric bilateral ophthalmopathy involving predominantly the left orbit and inferior rectus muscle. A CT scan of the brain was unremarkable.

Question 3
What is the diagnosis?

Question 4
What management options are available for this patient?

Answer 1
Diplopia is divided into monocular and binocular. Monocular diplopia is determined by the persistence of double vision when the unaffected eye is covered. The lesion can be localised to intraocular structures including refractive error, cataracts, lens dislocation and corneal opacity.
Binocular diplopia is determined by the absence of diplopia when either eye is covered. Causes include:

- Orbital (trauma, tumour, infection)
- Neurological
  - cranial nerve nucleus damage (infarct)
  - cranial nerve injury (CN III, IV, VI secondary to trauma, tumour, diabetes, sarcoidosis, vasculitis, aneurysm)
  - neuromuscular junction (myasthenia gravis)
  - muscle disorders (Graves’ ophthalmopathy).

**Answer 2**

First-line investigations in the general practice setting may include a full blood count, erythrocyte sedimentation rate (ESR), blood glucose and thyroid-stimulating hormone (TSH). Neuro-imaging such as CT or magnetic resonance imaging (MRI) of the brain or orbit may be required if there is clinical suspicion of a space-occupying lesion or neurological lesions such as multiple sclerosis. Further investigations such as electromyography (EMG), nerve conduction studies, tests for autoantibodies against acetylcholine receptors and follow-up investigations for any derangement in first-line tests may be ordered if indications exist.

**Answer 3**

Ophthalmopathy due to Graves’ hyperthyroidism.

**Answer 4**

Management of a patient with Graves’ ophthalmopathy should always first address the reversal of hyperthyroidism with antithyroid drug therapy, even though this has little-to-no effect in improving the ocular disease process. Prevention of corneal damage due to exposure includes artificial tears by day, ocular lubrication by night, punctal plugs, eyeshades and elevation of the bedhead at night. Severe or deteriorating orbitopathy may benefit from the anti-inflammatory properties of corticosteroids, and methylprednisolone is the drug of choice. Orbital radiation and surgical intervention may be required if medical treatment fails.

**Discussion**

Hyperthyroidism is a condition commonly encountered and managed in the general practice setting. The classical constellation of symptoms includes weight loss, increased appetite, sweating, tremor, anxiety, heat intolerance, palpitations, dyspnoea and diarrhoea. Depending on the cause, other manifestations may include the development of a goitre or ophthalmopathy unique to Graves’ disease.

Graves’ ophthalmopathy is an autoantibody-mediated process, whereby inflammation and fibrosis can occur and lead to an increase in volume of extra-ocular muscles and retro-orbital tissues. Patients may complain of eye irritation, retro-orbital pressure and pain, excessive tearing, blurriness, diplopia and visual loss. Signs of ophthalmopathy include proptosis, periocular oedema and restriction of ocular movements. Smoking is a major modifiable risk factor with an odds ratio of 7.7 when compared with non-smokers. The likely pathology involves an increase in the volume of connective tissue in the orbit rather than an effect on the extraocular muscles. The time course of Graves’ ophthalmopathy is variable, and is noted to precede any other symptom of hyperthyroidism in 20% of cases, in conjunction in 40% of cases and 40% following other symptoms.

In Murtagh’s General Practice, seven ‘masquerades’ are identified as diagnoses that are commonly missed; these include depression, diabetes, drugs, anaemia, thyroid disorder, spinal dysfunction and urinary tract infection. In line with Murtagh’s model, this case elucidates the importance of considering hyperthyroidism in patients presenting solely with diplopia. In essence, remembering to screen for masquerades, especially in the general practice setting, would save patients from unnecessary referrals and time spent within the medical system.

**Key points**

- Diplopia is divided into monocular and binocular.
- Monocular diplopia indicates pathology of intraocular structures.
- Binocular diplopia is usually caused by pathology of the orbit, cranial nerve palsy, neuromuscular junction disease, myopathy or systemic disease.
- Thyroid disease should be excluded in patients with binocular diplopia since Graves’ disease can present with ophthalmopathy as its only clinical feature.

**Authors**

Xiao Chen MBBS, Intern, The Alfred Hospital, Melbourne, VIC. cldxiao@hotmail.com
A masquerade presenting with diplopia

CLINICAL

Awaldeep Singh MBBS (Hons), Intern, The Alfred Hospital, Melbourne, VIC
Satpal Singh MBBS, PG, DipOccMed, MAFP, FRACGP, AM, General Practitioner, Rumbalara Aboriginal Medical Service, Mooroopna, VIC
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correspondence afp@racgp.org.au