Managing a patient with ptosis

A 50-year-old male patient is referred from the Accident & Emergency department with a history of ptosis. How will you manage this patient?

Clinical management

Clinical management starts with a sound clinical history.

Current history

The history should cover the following points:

- Unilateral or bilateral?
- The speed of onset – acute, sub-acute or chronic?
- The rate of progression. Are there any relieving or exasperating factors?
- History of trauma to the eyelid.
- History of trauma to the head, neck or chest.
- History of contact lens wear.
- History of allergic eye disease.
- Previous ophthalmic surgical history including corneal refractive surgery and blepharoplasty.
- History of smoking.

An old photograph is a very valuable tool in the assessment.

Other medical problems

- Any associated symptoms especially fatigueability, dysphagia or any muscle weakness.
- History of bleeding diathesis and haematologic problems.
- Features of over active or underactive thyroid.

An important part in the assessment of a patient with ptosis should involve excluding the causes for pseudoptosis. Some of the causes are:

- Contralateral eyelid retraction
- Post enucleation socket syndrome
- Aberrant reinnervation of the facial nerve
- Hemifacial spasm
- Brow ptosis.

Examination

Patients with ptosis should undergo a routine ophthalmic examination and neurological examination.

Thorough evaluation of the ptosis is essential, not only to select the appropriate surgical procedure but to also inform patients about the expected surgical outcome. Pre- and post- operative photographs are good clinical practice.

Before assessing the patient for the ptosis:

- Take a step back and assess the face for abnormal head posture.
- Assess the forehead for overacting frontalis, brow position, midface droop, and position of the angle of the mouth.

Ptosis examination

The assessment includes the following measurements:

- Palpebral aperture (normal range: male 8-10mm, female 9-12mm). Based on the difference in palpebral aperture measurement ptosis can be classified into mild, moderate or severe.
  Mild ptosis – 2mm
  Moderate ptosis – 3mm
  Severe ptosis – >4mm.
- Margin reflex distance 1 (MRD1). This is the distance between central corneal reflex and upper lid margin.
- Margin reflex distance 2 (MRD2). This is the distance between central corneal reflex and lower lid margin.
- Levator function is measured after stabilising the brow.
  Normal – >15mm
  Good – 12-14mm
  Fair – 8-11mm
  Poor – <8mm.
- Margin crease distance. This is the distance between upper lid margin and upper lid skin crease in down gaze. Can be absent in congenital ptosis and increased in aponeurotic disinsertion.
- Bell's phenomenon.
- Marcus Gunn phenomenon more important in congenital ptosis.
- Herings law: always document the effect of lifting the ptotic lid on the other eyelid.
- Extraocular movements.
- Facial muscle function.
- Phenylephrine test for mild ptosis.
  Compare MRD1 before and after instilling Phenylephrine and not palpable aperture.
- Lid contour – medial or lateral droop.
- Lash ptosis – document the presence of floppy eyelids.
- Evert the eyelid.

Mr Bertie Fernando,
MRCS FRCO,
East Lancashire Hospitals
NHS Trust, UK.

TAKE HOME MESSAGE

A good history, thorough examination and eversion of the eyelid are absolute essentials for accurate management.

A careful history and examination with attention to the measurements which characterise the ptosis are crucial in diagnosis. These include marginal reflex distance, levator function, orbicularis closure strength and importantly skin crease height (normal: Caucasian males 7-8mm and females 8-9mm).

Other neurological signs need to be noted too, including the presence of anisocoria and abnormal ocular movements.

Contrary to the commonly held belief of medical students, myasthenia and Horner's syndrome are not the commonest causes of acquired ptosis. By far, the commonest cause of acquired ptosis is aponeurotic dehiscence – often due to ageing or contact lens wear. Although most patients with aponeurotic dehiscence type ptosis give a good history of slow progressive worsening over several

years, many patients referred to the oculoplastic clinics only became aware of their ptosis after it had been pointed out to them, e.g. by their optician. Hence these patients then believe their ptosis is of acute onset. Indeed, it is only after looking at serial photographs that some patients realise their ptotic eyelids have been progressive in onset. Subtle changes in the configuration of the eyelid fold itself often precede the descent of the eyelid margin. In the early stages of aponeurotic dehiscence, the skin crease starts to elevate first and patients often notice increased exposure of the eyelid – so called 'upper lid show' as the lid fold rises. They may notice that upper lid sulcus becomes increasingly hollow (so called 'superior sulcus deformity') as the associated orbital fat retracts back into the socket with increasing dehiscence of the levator aponeurosis.

Levator aponeurotic dehiscence type ptosis, although commonly bilateral, can be quite asymmetrical in severity. It is characterised by good levator function (since the amplitude of contraction of the levator muscle remains unchanged) and an elevated skin crease height. Since the entire aponeurosis-tarsal plate apparatus (known as the posterior lamella) is effectively elongated, the ptosis is usually just as apparent when the patient looks down – so called 'downgaze ptosis'. This is in contrast to neurological causes of ptosis such as third nerve palsy, chronic progressive external ophthalmoplegia (CPEO) and myasthenia etc., where the levator aponeurosis-tarsal plate apparatus is essentially of normal length and thus in downgaze the eyelid no longer looks ptotic.

Signs for more sinister causes of acute onset ptosis include anisocoria (Horner's syndrome and third nerve palsy), ocular motility disturbance and / or reduced levator function (third nerve palsy, CPEO and myasthenia).

In Horner's syndrome, reduced tonus of the Muller's muscle can lead to elongation of the posterior lamella and hence elevation of the skin crease and downgaze ptosis. The ptosis is usually mild (<3mm) but can look like an aponeurotic type ptosis unless one actively looks for anisocoria. Apraclonidine and cocaine testing are sometimes necessary to confirm the diagnosis.

The ice test (where an ice pack is applied to both eyes for two to five minutes) is a sensitive and cheap test to look for ptosis secondary to myasthenia. (We commonly use ice cubes from the hospital kitchens in a pathology specimen bag.) Temporary reversal of the ptosis occurs in myasthenic patients, with a positive test being an increase in palpebral aperture of 2mm or more.

Mr David Cheung,
FRCOphth,
Consultant Ophthalmic Surgeon,
Russells Hall Hospital,
Dudley, UK.

SECTION EDITOR

Mr Yajati K Ghosh,
MBBS, FRCSEd Ophthal,
Consultant Ophthalmic Surgeon,
Birmingham &
Midland Eye Centre, UK.
E: yajati_ghosh@hotmail.com