Atopic Keratoconjunctivitis (AKC)

| Aetiology                                                                 | Severe ocular surface disease affecting some atopic individuals  
Complex immunopathology including T-cell mediated (type 4 hypersensitivity)  
Symptoms of AKC typically begin in the late teens or early twenties and can persist until the fourth or fifth decade of life. The peak incidence of AKC occurs between the ages of 30 and 50 years  
Sometimes follows childhood Vernal Keratoconjunctivitis (VKC) (see Clinical Management Guideline on Vernal Keratoconjunctivitis) |
|---|---|
| Predisposing factors                                                      | The majority of patients have a personal history of asthma, and eczema (atopic dermatitis)  
There may also be a family history of atopic disease  
Most patients have eczema affecting the eyelids and periorbital skin  
There is a strong association with staphylococcal lid margin disease  
Specific allergens may exacerbate the condition |
| Symptoms                                                                 | Ocular itching, burning, watering, usually bilateral  
Blurred vision, photophobia  
White stringy mucoid discharge  
Onset of ocular symptoms may occur several years after onset of atopy  
Symptoms usually year-round, with exacerbations |
| Signs                                                                     | Eyelids may be thickened, crusted and fissured  
Associated chronic staphylococcal blepharitis  
Tarsal conjunctiva: giant papillary hypertrophy, subepithelial scarring and shrinkage  
Entire conjunctiva hyperaemic  
Limbal inflammation  
Corneal involvement is common and may be sight-threatening: beginning with punctate epitheliopathy that may progress to macro-erosion, plaque formation (usually upper half), progressive corneal subepithelial scarring, neovascularisation, thinning, and rarely spontaneous perforation  
These patients are prone to develop herpes simplex keratitis (which may be bilateral), corneal ectasia such as keratoconus, atopic (anterior or posterior polar) cataracts, retinal detachment |
| Differential diagnosis                                                    | Vernal Keratoconjunctivitis  
Other allergic conjunctivitis, e.g. Giant Papillary Conjunctivitis (GPC) (often contact lens-related)  
Toxic Keratoconjunctivitis |

**Management by Optometrist**

- Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.
- **Non pharmacological**
  - Lid hygiene and treatment of associated staphylococcal blepharitis (see Clinical Management Guideline on Blepharitis)
  - Cool compresses
  - Advise avoidance of specific allergens if known, e.g. elimination of pets and carpeting, where necessary; instillation of air filtering devices and alterations to bedding materials (GRADE*: Level of evidence=low, Strength of recommendation=strong)
- **Pharmacological**
  - Systemic antihistamines e.g. cetirizine
  - Topical mast cell stabilisers, e.g. gutt. sodium cromoglicate 2%, gutt. lodoxamide 0.1%, or dual acting agents e.g. gutt olopatadine 0.1%, gutt

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Atopic Keratoconjunctivitis (AKC)
Version 14, Page 1 of 3
Date of search 23.11.18; Date of revision 26.11.18; Date of publication ab.cd.ef; Date for review 22.11.20
© College of Optometrists
### Atopic Keratoconjunctivitis (AKC)

<table>
<thead>
<tr>
<th>Management Category</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe corneal complications are common and potentially sight-threatening. If corneal epithelial macro-erosion or plaque are present:</td>
<td><strong>A3</strong>: First aid measures followed by urgent referral (within one week) to an ophthalmologist</td>
</tr>
<tr>
<td>Milder cases (without active corneal involvement):</td>
<td><strong>B1</strong>: Possible prescription of drugs; routine referral</td>
</tr>
</tbody>
</table>

**Possible management by Ophthalmologist**

- Often requires multidisciplinary management (ophthalmology, dermatology, immunology)
- Topical steroids with monitoring and management of complications, e.g. steroid glaucoma and cataract
- Topical/systemic antibiotics for lids
- Topical immunosuppression (e.g. ciclosporin, tacrolimus)
- Treatment of facial eczema and atopic blepharitis
- Surgery for atopic cataract

**Evidence base**

*GRADE*: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)

**Sources of evidence**

LAY SUMMARY

Atopic keratoconjunctivitis is a chronic (long-term) allergic condition of the eyelids and front surface of the eye. It is present in a high percentage of patients who have the skin condition, atopic dermatitis. Atopic keratoconjunctivitis requires long-term treatment to prevent sight-threatening complications such as scarring of the cornea (the clear window at the front of the eye). In the early stages of the disease, symptoms can be controlled by standard anti-allergy drugs. However, short-term use of steroid eye drops is often required when symptoms are severe. There is some evidence that cases that do not respond to steroids, or those requiring steroids eye drops long term, may benefit from ciclosporin or tacrolimus eye drops or ointment. Many cases need to be referred to the ophthalmologist for management, who in turn may refer them to specialists in other allergic diseases.
Aetiology
Commonest (80-90%) malignant tumour of the skin of white people
Commonest periocular malignancy
Incidence of BCC shows marked geographic variation; has been reported as 233 per 100,000 population per annum (in Wales) while in Australia incidence has been estimated as high as 884 per 100,000 per annum
Arises from the basal layer of the epidermis
Rarely metastasizes (incidence <0.1%)
Slow growing, locally invasive

Predisposing factors
Older patients (median age at diagnosis 67 years; rare under 40 years) M:F = 3:2
History of sunlight (UVB) exposure (particularly during childhood and adolescence)
Outdoor workers are at significantly increased risk
Fair complexion
History of previous BCC or other non-melanoma skin cancer
Immune compromise (e.g. HIV infection, immunosuppression)

Symptoms
Slow developing, non-resolving lesion of eyelid skin
Usually painless, may bleed

Signs
Location in order of prevalence (commonest first)
- lower lid
- medial canthus (deeper tissue penetration more likely with possible invasion of orbit or paranasal sinuses) (may be hidden by spectacle nose pad)
- upper lid
- lateral canthus

Three clinical presentations in order of prevalence (commonest first)
- Nodular (hard nodule, pearly appearance, abnormal (telangiectatic) vessels)
- Nodulo-ulcerative (as nodular but with raised rolled border surrounding central ulcer, may bleed)
- Morphoeic or sclerosing (flat hardened plaque of thickened skin, without surface vascularisation, ill-defined border making it difficult to determine area of involvement)

Occasional secondary infection or inflammation
- overlying purulent discharge or crusting

Change in lid contour/redirection or loss of eyelashes
Loss of texture of surrounding skin
Later cases sometimes pigmented (more prominent in dark skinned races)

Differential diagnosis
- Squamous cell carcinoma, chalazion, keratoacanthoma, actinic keratosis, molluscum contagiosum, papilloma
- Anterior marginal blepharitis can resemble sclerosing BCC
- BCC diagnosis is suspected clinically but is usually confirmed by histology which can also help to define the clinical subtype

Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

Non pharmacological
Document with photography if possible
Refer with details of location, size and history (to aid assessment of urgency)
Advise patient of possible diagnosis
Basal cell carcinoma (BCC) (periocular)

**Pharmacological**
None

**Management Category**
B1: no treatment by optometrist; routine referral
This slow growing neoplasia rarely metastasises but if left untreated could threaten sight (or life if BCC invades beyond orbit)

**Possible management by Ophthalmologist**

- Biopsy for histopathology analysis to confirm diagnosis
- Surgery and radiotherapy appear to be the most effective treatments with surgery showing the lowest failure rates (up to 98% success). Mohs micrographic surgery is considered the better alternative for treatment of certain types of periocular BCC because it has the highest chance of curing the disease and minimises the size of the defect that needs to be repaired. It has been reported to have the lowest recurrence rate of any treatment modality in a large prospective case series from Australia, although this has yet to be confirmed by RCTs
- Other treatment modalities that are sometimes recommended include cryotherapy, photodynamic therapy, carbon dioxide laser ablation, and chemotherapy including Imiquimod (topical immune response modulator)

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

**Sources of evidence**


**LAY SUMMARY**

Basal Cell Carcinoma (BCC) is a low-risk cancer that is the most common malignant tumour of the skin in white people. BCC rarely spreads to other parts of the body. Instead, it slowly enlarges, causing no pain, though bleeding may occur from the surface. Around the eye, the most usual form and location is a small hard whitish nodule that appears on the lower eyelid.

Figures from Wales indicate that BCC occurs in just under 0.25% of the population every year. In other parts of the world, especially where ultra-violet (UV) exposure is higher, more people are affected; for example, the figure may be three to four times higher in Australia. People who work outdoors are more likely to develop BCC.
It is very important to distinguish BCC from other growths that sometimes appear on the eyelids. It is usual to take a small sample of the tumour (known as a biopsy) for examination in the laboratory, so that the diagnosis can be confirmed and a treatment plan developed.

The usual treatment is either radiotherapy or surgery to remove the tumour. A special kind of surgery, known as Mohs micrographic surgery, is sometimes recommended. In this procedure, thin slices of the tumour are removed one after another and examined under the microscope until the surgeon is satisfied that all of the tumour has been removed. The result is complete removal of the tumour with the smallest possible wound.
# BLEPHARITIS (LID MARGIN DISEASE)

**Aetiology**

<table>
<thead>
<tr>
<th>Anterior blepharitis (also known as Anterior Lid Margin Disease)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- bacterial (usually staphylococcal)</td>
</tr>
<tr>
<td>- caused by (1) direct infection, (2) reaction to staphylococcal exotoxin or (3) allergic response to staphylococcal antigen</td>
</tr>
<tr>
<td>- seborrhoeic (disorder of the ciliary sebaceous glands of Zeis)</td>
</tr>
</tbody>
</table>

**Posterior blepharitis (also known as Posterior Lid Margin Disease)**

| - meibomian gland dysfunction (MGD) |
| - bacterial lipases break down meibomian lipids |
| - meibomian secretion becomes abnormal both chemically and physically |
| - tear film becomes unstable |

**Mixed anterior and posterior blepharitis**

| - elements of both conditions are present |

*All of these conditions are typically bilateral, and chronic or relapsing*  

**Dry Eye Disease**

- 50% of people with staphylococcal blepharitis  
- 25-40% of people with seborrhoeic blepharitis  

**Predisposing factors**

<table>
<thead>
<tr>
<th>Seborrhoeic blepharitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>- seborrhoeic dermatitis (for example, of the scalp)</td>
</tr>
</tbody>
</table>

**Demodex**

- *D folliculorum* is an ectoparasite that occurs normally in the lash follicles  
- *D brevis*, also an ectoparasite, is found in meibomian glands  

**Long-term contact lens wear**

**Ocular rosacea (a cause of posterior blepharitis)**

**Symptoms**

Blepharitis may be asymptomatic. However, when present, the symptoms of anterior blepharitis, posterior blepharitis and mixed anterior and posterior blepharitis are similar:

- ocular discomfort, soreness, burning, itching  
- mild photophobia  
- symptoms of dry eye including blurred vision and contact lens intolerance  

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## Signs

<table>
<thead>
<tr>
<th>Anterior blepharitis (staphylococcal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• lid margin hyperaemia</td>
</tr>
<tr>
<td>• lid margin swelling</td>
</tr>
<tr>
<td>• crusting of anterior lid margin (scales at bases of lashes)</td>
</tr>
<tr>
<td>• misdirection of lashes</td>
</tr>
<tr>
<td>• loss of lashes (madarosis)</td>
</tr>
<tr>
<td>• recurrent styes and (rarely) chalazia</td>
</tr>
<tr>
<td>• conjunctival hyperaemia</td>
</tr>
<tr>
<td>• secondary signs include: punctate epithelial erosion over lower third of cornea; marginal keratitis; phlyctenulosis; neovascularisation and pannus; mild papillary conjunctivitis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Anterior blepharitis (seborrhoeic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• lid margin hyperaemia</td>
</tr>
<tr>
<td>• oily or greasy deposits on lid margins</td>
</tr>
<tr>
<td>• conjunctival hyperaemia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Anterior blepharitis (Demodex)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• lid margin hyperaemia</td>
</tr>
<tr>
<td>• ‘cylindrical dandruff’: characteristic clear sleeve (collarette) covers base of lash, extending further up lash than flat staphylococcal rosettes</td>
</tr>
<tr>
<td>• persistent infestation of the lash follicles may lead to misalignment, trichiasis or madarosis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Posterior blepharitis (MGD is the most common cause)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• thick and/or opaque secretion at meibomian gland orifices, making it difficult or impossible to express oil by finger pressure</td>
</tr>
<tr>
<td>• foam in the lower tear film meniscus (due to excess tear film lipid)</td>
</tr>
<tr>
<td>• plugging of duct orifices with abnormal lipid leading to dilatation of glands and formation of microliths and chalazia</td>
</tr>
<tr>
<td>• conjunctival hyperaemia</td>
</tr>
<tr>
<td>• evaporative tear deficiency, unstable pre-corneal tear film</td>
</tr>
<tr>
<td>• secondary signs include: punctate epithelial erosion over lower third of cornea; marginal keratitis; scarring; neovascularisation and pannus; mild papillary conjunctivitis</td>
</tr>
</tbody>
</table>

## Differential diagnosis

<table>
<thead>
<tr>
<th>Allergy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermatoconjunctivitis medicamentosa (see Clinical Management Guideline on Conjunctivitis Medicamentosa)</td>
</tr>
<tr>
<td>Dacryocystitis</td>
</tr>
<tr>
<td>Parasitic infestation (e.g. Phthirus pubis infestation)</td>
</tr>
<tr>
<td>Preseptal cellulitis</td>
</tr>
<tr>
<td>Herpes (simplex or zoster)</td>
</tr>
<tr>
<td>Meibomian gland carcinoma (usually unilateral)</td>
</tr>
</tbody>
</table>

## Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

<table>
<thead>
<tr>
<th>Non pharmacological</th>
<th>Management of Dry Eye Disease, if also present: see Clinical Management Guideline on Dry Eye Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lid hygiene, consisting of lid cleansing using a variety of measures, is the first line of management regardless of type of blepharitis</td>
</tr>
<tr>
<td></td>
<td>Lid cleansing measures wipe away bacteria and deposits from lid margins and lead to improved signs and symptoms in the majority of individuals. However, there is insufficient high quality evidence on the comparative efficacy of the various lid hygiene regimes. There is evidence that long-term compliance with lid hygiene measures may be poor (GRADE*: Level of evidence = moderate, Strength of recommendation = strong)</td>
</tr>
</tbody>
</table>
| Wet warm compresses loosen collarettes and crusts in anterior blepharitis. Dry warm compresses melt meibum in posterior blepharitis (compress applied to lid skin twice daily for not less than 5 minutes at 40°C. Commercial products are available that are able to maintain temperatures in this region) (GRADE*: Level of evidence = weak, Strength of recommendation = strong) | Advise the avoidance of cosmetics, especially eye liner and mascara  
Advise patient to return/seek further help if symptoms persist  
Complete eradication of the blepharitis may not be possible, but long term compliance with these measures should reduce symptoms and minimise the number and severity of relapses |
## Pharmacological

Staphylococcal and seborrhoeic blepharitis may benefit from topical antibiotics if not controlled by first line management

- antibiotic ointment (e.g. chloramphenicol) twice daily; place in eyes or rub into lid margin with fingertip
- topical azithromycin (NB off-label use)

(GRADE*: Level of evidence = moderate, Strength of recommendation = weak)

In patients with posterior blepharitis, systemic antibiotics may be effective as a second line treatment

- consider prescribing a systemic tetracycline, such as oxytetracycline, doxycycline or minocycline (contraindicated in pregnancy, lactation and in children under 12 years; various adverse effects have been reported). Such treatment will need to be continued for several weeks or months and the dosage may need to be varied from time to time

(GRADE*: Level of evidence = low, Strength of recommendation = weak)

Consider Demodex blepharitis if characteristic ‘cylindrical dandruff’ is present at roots of eyelashes or if blepharitis is refractory to treatment. Demodex mites can be dose-dependently killed by weekly lid cleansing with 50% tea tree oil (see evidence base), but this should be undertaken only by experienced practitioners as such preparations are toxic to the ocular surface. Preparations containing 4-terpineol (an active ingredient of tea tree oil) are commercially available for patient use

(GRADE*: Level of evidence = low, Strength of recommendation = weak)

## Management Category

<table>
<thead>
<tr>
<th>Management Category</th>
<th>B2: alleviation/palliation: normally no referral</th>
<th>B1: initial management followed by routine referral if three months of pharmacological therapy does not produce sufficient response</th>
<th>A3: in unilateral cases, if meibomian gland carcinoma is suspected, refer urgently (within one week)</th>
</tr>
</thead>
</table>

## Possible management by Ophthalmologist

- Microbiological investigations including culture and sensitivity testing To minimise risk of post-operative infection, management of blepharitis prior to penetrating ocular surgery (e.g. trabeculectomy)
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**CLINICAL MANAGEMENT GUIDELINES**

**Blepharitis (Lid Margin Disease)**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*

**Sources of evidence**


Lindsley K, Matsumura S, Hafez E, Akpek EK. Interventions for chronic blepharitis. Cochrane Database of Systematic Reviews 2012;5:CD005556


LAY SUMMARY

Blepharitis is a condition in which chronic (i.e. long-term) inflammation of the eyelid margins causes symptoms of eye irritation. Sometimes there are no symptoms. There are two types of blepharitis, which sometimes occur together:

- Anterior blepharitis, which affects the outside front edge of the eyelids (near or among the roots of the eyelashes).
- Posterior blepharitis, which is most commonly caused by Meibomian Gland Dysfunction (MGD), results when the condition affects the inside rims of the eyelids (just behind the eyelashes) which contain the meibomian glands. (The meibomian glands produce a thin layer of oil which normally prevents the tears from evaporating too quickly; if they are inflamed, this mechanism does not work properly.)

Antibiotics in the form of eye drops or ointments (and in some cases antibiotics taken by mouth) can potentially lessen symptoms and are effective in clearing bacteria from the eyelid margins. Lid hygiene reduces symptoms for the majority of patients with either anterior or posterior blepharitis. However, there is no strong evidence that any of these treatments can completely cure the condition.
# Cellulitis, preseptal and orbital

## Aetiology

Infections of the periorbital and orbital tissues range in severity, from relatively minor to potentially life-threatening. These infections occur most commonly in children under the age of 10 years.

**Preseptal cellulitis**
- bacterial infection of tissues lying anterior to the orbital septum (therefore not an orbital condition)
- in young children, high risk of extension into the orbit

**Orbital cellulitis**
- bacterial infection of tissues lying posterior to the orbital septum (within the orbit)
- severe sight- and life-threatening emergency

For both conditions, the usual causative organisms are *Staphylococcus*, *Streptococcus* and *Haemophilus* species.

## Predisposing factors

**Preseptal cellulitis:**
- upper respiratory tract infection
- dacryocystitis
- hordeolum
- impetigo (skin infection)
- trauma, sharp or blunt, around eye
- recent surgery around eye

**Orbital cellulitis:**
- acute sinusitis (especially ethmoid sinusitis)
- trauma including orbital fracture
- dacryocystitis
- preseptal cellulitis
- dental abscess

## Symptoms

**Preseptal cellulitis:**
- acute onset of swelling, redness and tenderness of lids
- fever
- malaise
- irritability in children

**Orbital cellulitis:**
- sudden onset of unilateral swelling of conjunctiva and lids that may be painful
- pain on ocular movement
- blurred vision and reduced visual acuity
- diplopia
- fever
- severe malaise

## Signs

**Preseptal cellulitis:**
- erythema of skin (can extend beyond orbital rim)
- lid oedema, warmth, tenderness
- ptosis
- pyrexia (fever greater than 38°C, normal temperature ranges from 36-37.5°C)

**Orbital cellulitis:**
- proptosis
**Cellulitis, preseptal and orbital**

- restriction of extraocular motility
- pain with eye movement
- visual acuity may be reduced
- pupil reactions may be abnormal (RAPD)
- pyrexia

Distinguishing between preseptal cellulitis and orbital cellulitis can be difficult based on clinical observations alone (especially in children) although the following table may be helpful for differential diagnosis:

<table>
<thead>
<tr>
<th>Feature</th>
<th>Preseptal cellulitis</th>
<th>Orbital cellulitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proptosis</td>
<td>absent</td>
<td>present</td>
</tr>
<tr>
<td>Ocular motility</td>
<td>normal</td>
<td>painful, restricted</td>
</tr>
<tr>
<td>Visual acuity</td>
<td>normal</td>
<td>reduced in severe cases</td>
</tr>
<tr>
<td>Colour vision</td>
<td>normal</td>
<td>reduced in severe cases</td>
</tr>
<tr>
<td>RAPD</td>
<td>normal</td>
<td>present in severe cases</td>
</tr>
</tbody>
</table>

(Modified from a table in Denniston AKO and Murray PI: Oxford Handbook of Ophthalmology, 3rd edition, OUP 2014)

Contrast-enhanced CT scanning should be performed in all patients with symptoms and signs suggestive of orbital cellulitis

### Differential diagnosis

**Preseptal cellulitis:**
- orbital cellulitis
- hordeolum (external or internal)
- acute blepharitis
- viral conjunctivitis with eyelid swelling
- allergic conjunctivitis with eyelid swelling
- angioneurotic oedema (if bilateral): could indicate severe systemic allergic reaction, e.g. in peanut allergy
- insect bite or sting (if unilateral): look for skin lesion(s)

**Orbital cellulitis:**
- cavernous sinus thrombosis
- mucormycosis (fungal infection)
- sarcoidosis
- dysthyroid exophthalmos
- neoplasia with inflammation

### Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

| Non pharmacological | None |
| Pharmacological     | None |

**Management Category**

- **Preseptal and orbital cellulitis:**
  - A1: emergency (same day) referral to ophthalmologist or A&E
  - Department, no intervention

### Possible management by Ophthalmologist

Management of orbital infections typically involves a multi-disciplinary approach
Cellulitis, preseptal and orbital

Preseptal cellulitis:
- confirmation of diagnosis
- CT scan
- children may require admission to hospital for observation
- systemic antibiotics (oral and/or parenteral)

Orbital cellulitis:
- confirmation of diagnosis
- CT scan
- blood tests, possibly including microbial culture
- admission to hospital
- systemic antibiotics (intravenous)
- drainage of orbital abscess and microbiological culture of fluid
- co-management with ENT and paediatric specialist colleagues

Evidence base

Sources of evidence


LAY SUMMARY

Cellulitis means inflammation of the soft tissues, often due to infection. Preseptal and orbital cellulitis are infections of the soft tissues in the socket that surrounds the eye, usually caused by common bacteria. They may follow a cold, sinusitis, an infection of the eyelid such as a stye, an infection of the tear drainage channels, or injury or recent surgery near the eye. It is important to try to distinguish between these two forms of cellulitis. Preseptal cellulitis is usually mild, except in young children, but orbital cellulitis can result in generalised infection which can be a life-threatening emergency. All cases need emergency referral to the ophthalmologist or to an Accident and Emergency Department. Most will need to be admitted to hospital for tests and antibiotic treatment and a number of different specialists may be involved: ophthalmologists, ear, nose and throat specialists, and paediatricians (children’s doctors).
## Chalazion (Meibomian cyst)

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Blockage of Meibomian gland duct with retention and stagnation of secretion. May occur spontaneously or follow an acute hordeolum (internal).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Painless lid lump. Usually single; sometimes multiple. May be recurrent. May rupture through the skin. (Sometimes) blurred vision from induced astigmatism.</td>
</tr>
<tr>
<td>Signs</td>
<td>Well-defined, 2-8mm diameter subcutaneous nodule in tarsal plate. Lid eversion may show external conjunctival granuloma. Induced astigmatism/hyperopia may cause change in refraction. May be associated blepharitis.</td>
</tr>
<tr>
<td>Differential diagnosis</td>
<td>Hordeolum (external or internal). Sebaceous cyst of skin. Meibomian gland carcinoma (consider if lesion recurrent).</td>
</tr>
</tbody>
</table>

### Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.

**Non pharmacological**

Between 21% and 80% resolve on conservative management (warm compresses and lid massage; may take weeks or months).

If persistent, large, recurrent or causing corneal distortion then refer for management by ophthalmologist.

Regular lid hygiene for blepharitis (see Clinical Management Guideline on Blepharitis).

(GRADE*: Level of evidence=low; Strength of recommendation=strong)

**Pharmacological**

None (but see Clinical Management Guideline on Hordeolum [internal])

**Management Category**

- B2: alleviation/palliation: normally no referral
- B1: routine referral to ophthalmologist if persistent or recurrent, if causing significant astigmatism or if cosmetically unacceptable

### Possible management by Ophthalmologist

- Incision and curettage where appropriate.
- Intra-lesion injection of steroid (may be preferred in children).
- Trials have indicated uncertainty as to the relative benefits of intralresional triamcinolone injection compared with incision and curettage.

### Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see [http://www.gradeworkinggroup.org/index.htm](http://www.gradeworkinggroup.org/index.htm))

**Sources of evidence**

Chalazion (Meibomian cyst)

<table>
<thead>
<tr>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perry HD, Serniuk RA. Conservative treatment of chalazia Ophthalmology 1980;87(3):218-21</td>
</tr>
</tbody>
</table>

LAY SUMMARY

A chalazion, also known as a Meibomian cyst, is a common condition of the eyelid caused by blockage of the openings of the oil-producing Meibomian glands. It is usually felt as a small firm lump in the upper or lower eyelid. The condition often gets better with a combination of warm compresses and massage. However if it does not settle, it can be treated by a steroid injection or the cyst can be removed by a minor surgical procedure.
### Aetiology

Multifactoral aetiology not fully understood  
Type I immediate hypersensitivity mediated by IgE  
- possible antigens:  
  - altered host protein on lens surface  
  - bacterial cell wall constituents  
  - other lens contaminants  
- reaction causes degranulation of mast cells  
- products of degranulation stimulate recruitment of basophils and eosinophils to conjunctival epithelium  
Type IV delayed hypersensitivity mediated by T-cells  
- amplifies the inflammatory response  

Trauma to tarsal conjunctival surface releases neutrophil chemotactic factor  
- sources of trauma  
  - contact lenses  
  - ocular prostheses  
  - protruding sutures, extruding scleral buckles, filtration blebs  
  - elevated corneal deposits

### Predisposing factors

- More common in soft compared to rigid lenses  
  - occurs in silicone hydrogel, as well as hydrogel, lens wearers  
  - lens deposits  
  - thick or poorly designed or manufactured lens edges  
  - meibomian gland dysfunction  
  - atopy

### Symptoms

- Itching and non-specific irritation  
  - may increase after lens removal (manipulation of lids mechanically stimulates mast cell degranulation with release of vasoactive substances including histamine)  
- Mucus discharge  
- Increased lens movement  
- Loss of lens tolerance  
- Decreasing comfort (may abandon wear)  
- Blurred vision  
  (NB: poor correlation of severity with symptoms and signs)

### Signs

- Almost always bilateral  
- Upper tarsal conjunctiva (lower usually not affected)  
  - papillae  
    - macropapillae (diameter between 0.3 and 1 mm) or giant papillae (diameter > 1 mm)  
    - apices of papillae may stain with fluorescein when inflammation active  
    - apices may be whitish due to scarring in chronic cases  
  - hyperaemia  
  - stringy mucus in tear film and on conjunctival surfaces  
  - conjunctival oedema

### Differential diagnosis

Vernal Keratoconjunctivitis, Atopic Keratoconjunctivitis, Seasonal Allergic
### Conjunctivitis, Superior Limbic Keratoconjunctivitis
- Contact lens history will aid diagnosis

### Distinguish papillae from follicles:

<table>
<thead>
<tr>
<th>Follicles</th>
<th>Papillae</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Hyperplasia of lymphoid tissue</td>
<td>- Hyperplasia of epithelium</td>
</tr>
<tr>
<td>- Generally seen in viral or chlamydial conditions</td>
<td>- Usually more discrete and more red than follicles</td>
</tr>
<tr>
<td>- Smooth, pale, pink-to-yellow, elevated lesions</td>
<td>- Side walls of papillae appear perpendicular to tarsal plate</td>
</tr>
<tr>
<td>- Surrounded by displaced vessels</td>
<td>- Contain vascular core visible at apex as vascular tuft</td>
</tr>
</tbody>
</table>

### Management by Optometrist

**Non pharmacological**
- **Removal of lens deposits**
  - Replace soft lenses more frequently
  - Improve hygiene – more rigorous surfactant cleaning, more frequent enzyme use
  - Polish or replace rigid lenses

**Reduce exposure time**
- Abandon extended wear
- Reduce daily wearing time to minimum possible
- Cease wear for a period in some cases

**Optimise lens fit, material and wearing regime**
- Rigid lens: alter overall diameter (repositions lens edge relative to tarsus), reduce edge clearance and edge thickness
- Soft lens: change material to one with improved deposit resistance, and/or lower modulus, and/or change edge profile
- Change to daily disposable soft lenses

**Ocular prostheses**
- Polish, adjust or replace prosthesis

*GRADE*: Level of evidence=low; Strength of recommendation=strong

### Pharmacological

**Topical mast cell stabilisers** (gutt. sodium cromoglicate 2%, gutt. lodoxamide 0.1%, gutt. nedocromil sodium 2%):
- Can be used while lens wear continues but preserved drops should not be instilled with soft lenses *in situ*
- Nedocromil sodium is yellow and may discolour soft lenses

*GRADE*: Level of evidence=low; Strength of recommendation=strong

**Topical combined anti-histamine/mast cell stabilizer** e.g. gutt. olopatadine 0.1% (off-licence use)

*GRADE*: Level of evidence=low; Strength of recommendation=strong

In cases that do not respond to other treatment, consider a six-week
**CL-associated Papillary Conjunctivitis (CLAPC), Giant Papillary Conjunctivitis (GPC)**

<table>
<thead>
<tr>
<th>Management Category</th>
<th>B3: management to resolution (normally no referral)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Possible management by Ophthalmologist</strong></td>
<td>A range of topical steroids in recalcitrant cases that do not respond to other treatment, especially where contact lens wear is medically indicated</td>
</tr>
</tbody>
</table>

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)

**Sources of evidence**


Bailey CS, Buckley RJ. Nedocromil sodium in contact lens-associated papillary conjunctivitis. Eye 1993;7(suppl):29-33


Khurana S, Sharma N, Agarwal T, Chawla B, Velpandian T, Tandon R, Titiyal JS. Comparison of olopatadine and fluorometholone in contact lens-induced papillary conjunctivitis. Eye Contact Lens 2010;36:210-4


**LAY SUMMARY**

Contact lens-associated papillary conjunctivitis (CLAPC) is an inflammatory condition affecting the transparent membrane which lines the back of the upper eyelid (tarsal conjunctiva). It can occur in people wearing soft or rigid contact lenses or an ocular prosthesis (artificial eye). People suffering from this condition experience eye irritation, which may lead them to abandon contact lens wear.
CL-associated Papillary Conjunctivitis (CLAPC),
Giant Papillary Conjunctivitis (GPC)

The vision may be blurred intermittently. The eyes are often red and the underside of the upper lid shows minute cobblestone-like swellings called papillae.

Treatment for CLAPC initially consists of improving contact lens hygiene and replacing lenses more frequently. Eye drops such as anti-histamines or mast cell stabilisers are often required to relieve symptoms and improve clinical signs. In more severe cases it may be necessary to use steroid eye drops for short periods.
### Aetiology
Conjunctival epithelial inclusion cysts containing epithelial and keratin debris, sometimes with secondary calcification.

### Predisposing factors
- Common, especially over the age of 50 years (prevalence in ophthalmology outpatient population has been reported as approximately 40%)
- Chronic conjunctivitis (any cause, including contact lens wear)
- Accumulation of lipid under conjunctiva, e.g. concretions grouped around an old chalazion

### Symptoms
- Usually none
- May erode through the epithelium
  - foreign body sensation

### Signs
- Small white/yellow-white bodies with distinct edges in tarsal (upper or lower) conjunctiva
- Single or multiple
- Usually <1mm diam, sometimes up to 3mm
  - appear larger if confluent
- Usually low profile but may be raised if large

### Differential diagnosis
- Conjunctival retention cysts
  - thin walled cysts containing clear or translucent fluid
- Follicles
  - focal lymphoid hyperplasia

## Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.

### Non pharmacological
- Treatment rarely required
- Artificial tears and lubricating ointments (drops for use during the day, unmedicated ointment for use at bedtime)
  - (GRADE*: Level of evidence = low, Strength of recommendation = strong)

  NB Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Clinical Management Guideline on Conjunctivitis Medicamentosa). They should be switched to unpreserved preparations.

- Eroded concretions leading to irritation can be removed at the slit lamp
  - topical anaesthetic
  - tease out with sterile hypodermic needle
  - any bleeding should respond quickly to finger pressure on the lid
    - (N.B. check first that patient has no bleeding disorder and is not taking aspirin or anti-coagulants)
  - consider topical antibiotic as prophylactic if infection seems likely (e.g. gutt. or oc. chloramphenicol)
  - (GRADE*: Level of evidence = low, Strength of recommendation = weak)

### Pharmacological
- No specific drug treatment available
- Topical anaesthetic and antibiotic for minor surgery as above
  - (GRADE*: Level of evidence = low, Strength of recommendation = weak)

## Management Category
B3: Management to resolution

## Possible management by Ophthalmologist
Not normally required

## Evidence base
*GRADE: Grading of Recommendations Assessment, Development and
LAY SUMMARY

Concretions are small white or yellowish dots, usually less than 1mm in diameter, commonly seen on the undersides of the eyelids. They contain cell debris and calcium. They may be the result of past inflammation. Occasionally they cause irritation or the feeling that there is something in the eye.

If concretions are causing symptoms, the optometrist may offer to remove them. After numbing the eye surface with an anaesthetic drop, the concretions can usually be teased out with the tip of a hypodermic needle. Rarely, antibiotic drops may be prescribed.

Such cases do not usually need to be referred to the ophthalmologist.
### Conjunctivitis (Acute Allergic)

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>A self-limiting urticarial reaction to an allergen (often unidentified) that comes into contact with the conjunctiva provoking an immediate (Type I) IgE-mediated response. Common in children. Allergens include: grass pollen, animal dander.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predisposing factors</td>
<td>History of allergic disease; can also occur without such history.</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Sudden eyelid swelling. Ocular itching. May be unilateral (if a direct contact response).</td>
</tr>
<tr>
<td>Signs</td>
<td>Lid oedema and erythema. Conjunctival chemosis (oedema): may bulge over lid margin or limbus. Watery or mucoid discharge (mild). Usually no papillae. No corneal involvement.</td>
</tr>
<tr>
<td>Differential diagnosis</td>
<td>Seasonal allergic conjunctivitis. Chemical trauma. Preseptal or orbital cellulitis.</td>
</tr>
</tbody>
</table>

**Management by Optometrist**

**Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.**

**Non pharmacological**

- Reassure patient: most cases resolve spontaneously within a few hours.
- Advise against eye rubbing (causes mechanical mast cell degranulation).
- Cool compresses may give symptomatic relief. (GRADE*: Level of evidence=low, Strength of recommendation=strong)
- If possible identify allergen and advise future avoidance.
- Advise patient to return/seek further help if symptoms persist.

**Pharmacological**

- Not normally required (although ocular lubricant drops and/or topical anti-histamines may provide symptomatic relief). (GRADE*: Level of evidence=low, Strength of recommendation=strong)
- If condition recurrent, prescribe prophylactic topical mast cell stabiliser, e.g. gutt. sodium cromoglicate 2% (as POM) or gutt. lodoxamide 0.1%, or dual-acting antihistamine/mast cell stabiliser, e.g. gutt. olopatadine 0.1% (off-licence use), or gutt. ketotifen 0.025% (off-licence use); maximum duration 4 months. (GRADE*: Level of evidence=low, Strength of recommendation=weak)

**Management Category** **B2: Alleviation/palliation – normally no referral**

**Possible management by Ophthalmologist**

- Not normally referred.

**Evidence base**

- *GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)*
- **Sources of evidence**
- Buckley RJ. Allergic eye disease – a clinical challenge. Clin Exp Allergy
LAY SUMMARY

Acute allergic conjunctivitis is an allergic reaction of the eyes, which causes a sudden swelling and redness of the eyelids and conjunctiva (the membrane covering the white of the eye), often associated with itching. It usually occurs in susceptible individuals, typically following exposure to grass pollen or animal fur. Most cases get better within a few hours without the need for drug treatment. However, anti-allergy eye drops may help to control symptoms in the short term and in people with recurrent episodes.
### Conjunctivitis (bacterial)

| Aetiology | Self-limiting bacterial infection of the conjunctiva, typically by:  
| --- | ---  
|  | • *Staphylococcus* species  
|  | • *Streptococcus pneumoniae*  
|  | • *Haemophilus influenzae*  
|  | • *Moraxella catarrhalis* |

| Predisposing factors | Children and the elderly have an increased risk of infective conjunctivitis  
| --- | ---  
|  | *(NB Bacterial conjunctivitis in the first month of life is a serious condition that must be referred urgently to the ophthalmologist. See Clinical Management Guideline on Ophthalmia Neonatorum)*  
|  | • contamination of the conjunctival surface  
|  | • superficial trauma  
|  | • contact lens wear *(NB infection may be Gram –ve)*  
|  | • secondary to viral conjunctivitis  
|  | • recent cold, upper respiratory tract infection *(NB refer also to Clinical Management Guideline on Conjunctivitis (viral, non-herpetic))* or sinusitis  
|  | • diabetes *(or other disease compromising the immune system)*  
|  | • steroids *(systemic or topical, compromising ocular resistance to infection)*  
|  | • blepharitis *(or other chronic ocular inflammation)* |

| Symptoms | Acute onset of:  
| --- | ---  
|  | • redness  
|  | • discomfort, usually described as burning or grittiness  
|  | • discharge *(may cause temporary blurring of vision)*  
|  | • crusting of lids *(often stuck together after sleep and may have to be bathed open)*  
|  | Usually bilateral – one eye may be affected before the other *(by one or two days)* |

| Signs | • lid crusting  
| --- | ---  
|  | • purulent or mucopurulent discharge  
|  | • conjunctival hyperaemia – maximal in fornices  
|  | • tarsal conjunctiva may show mild papillary reaction  
|  | • cornea: usually no involvement *(occasionally punctate epitheliopathy – mainly in lower third of cornea). If cornea significantly involved, consider possibility of gonococcal infection*  
|  | *(pre-auricular lymphadenopathy: usually absent)* |

| Differential diagnosis | Other forms of conjunctivitis  
| --- | ---  
|  | • epidemic keratoconjunctivitis *(e.g. adenovirus)*  
|  | • *Herpes simplex or Herpes zoster*  
|  | • Chlamydial infection  
|  | • allergy  
|  | Other causes of acute red eye  
|  | • angle closure glaucoma  
|  | • infective keratitis  
|  | • anterior uveitis |

### Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

| Non- | Often resolves in 5-7 days without treatment |
**Conjunctivitis (bacterial)**

<table>
<thead>
<tr>
<th>Pharmacological</th>
<th>Bathe/clean the eyelids with proprietary sterile wipes, lint or cotton wool dipped in sterile saline or boiled (cooled) water to remove crusting (GRADE*: Level of evidence = low, Strength of recommendation = strong)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharmacological</td>
<td>Advise patient that condition is contagious (do not share towels, etc.) Public Health England guidance states that school or nursery exclusion is not required for children with this condition</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>Treatment with topical antibiotic may improve short-term outcome and render patient less infectious to others (GRADE*: Level of evidence = high, Strength of recommendation = strong). Alternatives include: chloramphenicol 0.5% eye drops, chloramphenicol 1% ointment, azithromycin 1.5% eye drops, fusidic acid 1% viscous eye drops (NB high cost and narrower spectrum of activity than chloramphenicol) This recommendation is based on the conclusions of a Cochrane Review (Sheikh and Hurwitz 2012) which included trials conducted in primary and secondary care. However, an individual patient meta-analysis of studies exclusively based in primary care (Jefferis et al 2011) found only a marginal benefit of antibiotics over placebo. Patients with purulent discharge or a mild severity of red eye were found to benefit most from treatment with antibiotics Contact lens wearers with a diagnosis of bacterial conjunctivitis should be treated with a topical antibiotic effective against Gram -ve organisms, e.g. a quinolone such as levofloxacin or moxifloxacin, or an aminoglycoside such as gentamicin. Contact lenses should not be worn during the treatment period (GRADE*: Level of evidence = low, Strength of recommendation = strong). Advise patient to return/seek further help if symptoms persist beyond 7 days</td>
</tr>
<tr>
<td>Management Category</td>
<td>B3: Management to resolution Refer if condition fails to resolve, or if there is corneal involvement</td>
</tr>
<tr>
<td>Possible management by Ophthalmologist</td>
<td>If resistant to treatment, or recurrent, • conjunctival swabs taken for microscopy and culture and/or PCR analysis • treatment with other antibiotics, based on culture results</td>
</tr>
</tbody>
</table>

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)*

**Sources of evidence**

<table>
<thead>
<tr>
<th>ol_in%20schools_poster.pdf</th>
</tr>
</thead>
</table>

LAY SUMMARY

Acute bacterial conjunctivitis is an infection of the eye in which one or both eyes become red with associated discomfort. The condition is not normally serious and in most cases clears up without treatment. People with acute bacterial conjunctivitis are often given antibiotics, usually in the form of eye drops or ointment, to speed recovery. However, the benefits of antibiotics for the treatment of acute bacterial conjunctivitis have been questioned. Evidence from clinical trials in GP practices suggests that antibiotic drops may be less helpful, since these patients tend to have a less severe form of conjunctivitis than patients who are referred to a hospital eye clinic.
### Aetiology
The microorganism *Chlamydia trachomatis* is an obligate intracellular parasite (i.e. it cannot replicate outside a host cell)
- Serotypes A-C cause trachoma, the leading cause of infectious blindness worldwide
- Serotypes D-K are responsible for Ophthalmia Neonatorum (see Clinical Management Guideline on Ophthalmia Neonatorum) and adult inclusion conjunctivitis (this Guideline) (named for the intracytoplasmic [inclusion] bodies seen in Giemsa-stained conjunctival scrapings)
- many patients (up to 70%) have a concurrent genital infection (may be asymptomatic)

### Predisposing factors
Most common in young adulthood (15-35 years)
Sexual activity leading to genital infection with *C. trachomatis*:
- urethritis, cervicitis, prostatitis, proctitis (inflammation of the rectum), etc.; (may be asymptomatic)
- increased prevalence of Chlamydial conjunctival infection in sexual partner(s) which may be asymptomatic

### Symptoms
History usually more than two weeks
Ocular gritty sensation and sticky discharge
Drooping upper lid(s)
(the majority of cases are unilateral)

### Signs
**Lid and other features**
- oedema +/- ptosis (‘mechanical’)
- non-tender pre-auricular lymph node swelling (may or may not be present)
**Conjunctival features**
- hyperaemia and chemosis
- mucopurulent conjunctivitis
- large follicles in upper and lower fornices (double eversion of lid needed to view upper fornix)
- limbal and/or bulbar follicles may also be present
**Corneal features**
- epithelial keratitis, usually superior
- subepithelial infiltrates, similar to those seen in adenovirus KC
- marginal infiltrates
- superior pannus
(NB signs may be unilateral or asymmetrical or bilateral)

### Differential diagnosis
Other causes of acute red eye such as adenovirus keratoconjunctivitis, HSK, molluscum contagiosum

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

| Non pharmacological | Advise against contact lens wear  
|                      | (GRADE*: Level of evidence=low, Strength of recommendation=strong) |
| Pharmacological      | Symptomatic relief with ocular lubricants  
|                      | (NB even if diagnosis appears beyond doubt, do not commence specific treatment before referral as other STDs may also be present)  
|                      | (GRADE*: Level of evidence=low, Strength of recommendation=strong) |

**Management Category**
A3: first aid measures and urgent referral to ophthalmologist or GP with
**Conjunctivitis, Chlamydial (adult inclusion conjunctivitis)**

<table>
<thead>
<tr>
<th>Possible management by Ophthalmologist</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laboratory testing to confirm diagnosis</td>
</tr>
<tr>
<td>Liaison with Genito-Urinary Clinic, which will exclude other STDs and advise on treatment of patient and partner(s).</td>
</tr>
<tr>
<td>Treatment with systemic antibiotics (e.g. single dose of azithromycin or a short course of doxycycline). Patients and sexual partners must be treated</td>
</tr>
</tbody>
</table>

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see [http://gradeworkinggroup.org/toolbox/index.htm](http://gradeworkinggroup.org/toolbox/index.htm))*

**Sources of evidence**


**LAY SUMMARY**

Chlamydia is one of the most common sexually transmitted infections (STIs) in the UK. In the eyes, chlamydia can cause irritation, pain, swelling and discharge, usually affecting one eye only. Most patients presenting with chlamydial conjunctivitis will have an associated genital infection (of which they may be unaware). It is important that these patients are referred to genito-urinary medicine clinics for a full STI investigation. Chlamydial infection is usually treated with antibiotics, which can be very effective. Most people with chlamydial infection will be cured if they take their antibiotics correctly.
### Aetiology

Type I hypersensitivity reaction to specific airborne allergens. Conjunctival mast cell degranulation liberates histamine and other inflammatory mediators into the tissues and tear film, causing dilatation of conjunctival vessels (→red eye), increased permeability of blood vessels (→oedema), itch

#### Seasonal allergic conjunctivitis (hay fever conjunctivitis) (SAC)
- caused by seasonal allergens, especially grass pollen
- onset of symptoms associated with seasonal production of allergens, e.g. tree pollen: spring; grasses: early summer; weeds and fungal spores: late summer
- condition not sight-threatening, but may be damaging to quality of life and associated with a significant economic burden

#### Perennial allergic conjunctivitis (PAC)
- caused by non-seasonal allergens such as house dust mite or animal dander
- symptoms throughout the year; may be seasonal exacerbations
- less common and usually less severe than seasonal type

### Predisposing factors

Atopic disposition (40% of population of which only around half manifest allergic disease)
- Personal history of allergic disease (hay fever, asthma, eczema, food or drug allergy)
- Family history of allergic disease
- Exposure to allergens

### Symptoms

Red eye
- Itching of eye (main symptom)
- Watering of eye
- May be associated with sneezing and watery nasal discharge

#### SAC:
- symptoms seasonal with climatic variations

#### PAC:
- symptoms perennial but variable; seasonal exacerbations may occur

### Signs

- Lids: mild to moderate oedema (peri-orbital oedema in severe cases)
- Bulbar and tarsal conjunctiva: chemosis (oedema), hyperaemia and diffuse papillary reaction
- Cornea: uninvolved

### Differential diagnosis

- Vernal or Atopic Keratoconjunctivitis (cornea usually involved)
- Other allergic conjunctivitis
  - Acute Allergic Conjunctivitis (AAC) (see Clinical Management Guideline on Conjunctivitis (acute allergic))
  - contact conjunctivitis (e.g. to drug or preservative in eye drops) (see Clinical Management Guideline on Conjunctivitis medicamentosa)
  - Contact Lens-Associated Papillary Conjunctivitis (CLAPC), also known as Giant Papillary Conjunctivitis (GPC) (response to contact lens, suture, etc.) (see Clinical Management Guideline on Contact Lens-Associated Papillary Conjunctivitis [CLAPC])

### Management by Optometrist

**Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere**

**Non pharmacological**
- Identify allergen(s)
- Advise avoidance of allergen(s)
# Seasonal Allergic Conjunctivitis (Rhinoconjunctivitis, Hay Fever Conjunctivitis); Perennial Allergic Conjunctivitis

## Pharmacological

<table>
<thead>
<tr>
<th>Cool compresses for symptomatic relief</th>
<th>Advise against eye rubbing (causes mechanical mast cell degranulation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ocular lubricants for symptomatic relief</td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

Various topical treatment options are available but there is insufficient evidence to recommend the use of one type of medication over another; however, the choice of drug may be determined by compliance, cost, and availability of preservative-free formulation (if required). The twice daily dosing regime of dual-action antihistamines may be beneficial in contact lens wearers and in school-age children.

- topical mast cell stabilizers, e.g. sodium cromoglicate, lodoxamide
- topical antihistamine e.g. antazoline
- topical antihistamine + mast cell inhibitor, e.g. olopatadine or ketotifen
- topical NSAID, e.g. diclofenac sodium

Systemic antihistamine (e.g. tabs cetirizine or loratadine once daily)

- effective also for other symptoms of hay fever, e.g. allergic rhinitis

## Management Category

| B2: alleviation or palliation; normally no referral |
| B1: if conventional therapy fails, consider referral to Clinical Immunologist for consideration of sub-lingual or other form of immunotherapy |

## Possible management by Ophthalmologist

(Not normally referred)

## Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)

**Sources of evidence**


- Castillo M, Scott NW, Mustafa MZ, Mustafa MS, Azuara-Blanco A. Topical antihistamines and mast cell stabilisers for treating seasonal and perennial allergic conjunctivitis. Cochrane Database Syst Rev. 2015;6:CD009566

LAY SUMMARY
Seasonal Allergic Conjunctivitis (SAC) is the eye component of hay fever and one of the most common eye problems, affecting about one fifth of adults. It is caused when a substance called an allergen reaches the eye surface and sets off an allergic reaction. Allergens are usually airborne. Grass pollen is the most common of these and is at its most concentrated in June and July. The allergic reaction releases histamine into the tears and the surface tissues of the eye, causing redness and swelling of the conjunctiva (the membrane covering the white of the eye), watering and itching. People with SAC often have allergic symptoms affecting the nose, throat and sinuses, and they may have asthma, eczema and food or drug allergy also. SAC can be unpleasant and cause people to lose work or school days, but it does not damage the sight. It can be treated with anti-allergy drops or antihistamines in eye drop form. Antihistamine tablets can also be helpful, and will usually control hay fever also.
Perennial Allergic Conjunctivitis (PAC) is rarer than SAC but produces similar symptoms. The main difference is that it is a reaction to a year-round allergen, such as house dust mite. Its treatment is similar.


### Conjunctivitis (viral, non-herpetic)

| Aetiology | Adenovirus (more than 30 serotypes)  
|-----------|----------------------------------|  
|           | • commonest form of acute infective conjunctivitis  
|           | • spectrum of disease varies from mild to severe  
|           | • two syndromes of adenoviral infection:  
|           |   - pharyngoconjunctival fever (not dealt with in this Guideline)  
|           |   - epidemic conjunctivitis and keratoconjunctivitis (this Guideline)  
|           | Enterovirus 70 (EV70) and Coxsackievirus A24 (CA24v)  
|           | • acute haemorrhagic conjunctivitis (rare epidemics)  
| Predisposing factors | Recent cold or other upper respiratory tract infection  
|                     | Low standards of hygiene  
|                     | Crowded conditions (schools, camps, clinics)  
|                     | Eye clinics (transmission by clinicians’ fingers, tonometer prisms, etc.)  
| Symptoms | Acute onset  
|          | • redness  
|          | • discomfort, usually described as burning or grittiness  
|          | • watering  
|          | Eyelids may be stuck together in the morning and have to be bathed open  
|          | Often unilateral at first, becoming bilateral, first eye usually more affected  
|          | Blurred vision if central cornea involved  
|          | Systemic malaise  
| Signs | Watery discharge  
|       | Conjunctival hyperaemia (may be intense)  
|       | Follicles on palpebral conjunctiva, especially upper and lower fornix (if abundant, follicles can produce folds)  
|       | Petechial (pin-point) subconjunctival haemorrhages  
|       | Pseudomembranes on tarsal conjunctival surfaces (severe cases only)  
|       | Pre-auricular lymphadenopathy which may be tender (not present in every case)  
|       | Corneal involvement in some cases:  
|       |   • punctate epithelial lesions within first two weeks  
|       |   • later replaced by sub-epithelial lesions which may persist for months  
| Differential diagnosis | Other forms of conjunctivitis  
|                       | • bacterial  
|                       | • chlamydial  
|                       | • herpetic (simplex or zoster)  
|                       | • allergic  
|                       | Other causes of acute red eye  
|                       | • angle closure glaucoma  
|                       | • keratitis  
|                       | • anterior uveitis  
|                       | A point of care diagnostic test (AdenoPlus) is available (see NICE Medtech innovation briefing: https://nice.org.uk/guidance/mib46)  

**Management by Optometrist**

Practitioners should recognise their limitations and where necessary seek further advice or refer.
**Conjunctivitis (viral, non-herpetic)**

<table>
<thead>
<tr>
<th></th>
<th>Non pharmacological</th>
<th>Pharmacological</th>
<th>Management Category</th>
<th>Possible management by Ophthalmologist</th>
<th>Evidence base</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wash hands carefully before and after examination and clean equipment before next patient. Do not applanate with a re-usable tonometer prism as condition is highly contagious. Advise patient: • condition is normally self-limiting, resolving within one to two weeks • condition is highly contagious for family, friends and work colleagues (do not share towels, etc) • infection with adenovirus necessitates 2 weeks off work or school • cold compresses may give symptomatic relief • discontinue contact lens wear in acute phase. Review to monitor for painful or sight-compromising corneal involvement or development of conjunctival pseudomembrane (in either case, refer to ophthalmologist) (GRADE*: Level of evidence=low, Strength of recommendation=strong).</td>
<td>Antibacterial agents are not effective in viral conditions. Current topical and systemic anti-viral agents also ineffective in adenovirus infection. Artificial tears and lubricating ointments (drops for use during the day, unmedicated ointment for use at bedtime) may relieve symptoms. Topical antihistamines may be used for severe itching (GRADE*: Level of evidence=low, Strength of recommendation=strong).</td>
<td>B2: alleviation/palliation; normally no referral A2: first aid measures and emergency referral (same day) if conjunctivitis severe (e.g. presence of pseudomembrane) or if significant keratitis present (e.g. severe pain and/or visual loss).</td>
<td>Conjunctival swabs for virus isolation and strain identification. Currently available anti-viral medication is ineffective. Topical low dose steroids may be prescribed where sub-epithelial opacities affect vision but this may encourage long-term steroid dependency and is not supported by the evidence base. Topical steroid may also be indicated in the acute phase where there is conjunctival pseudomembrane formation.</td>
<td>*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see <a href="http://gradeworkinggroup.org/toolbox/index.htm">http://gradeworkinggroup.org/toolbox/index.htm</a>) Sources of evidence Azari AA, Barney NP. Conjunctivitis: a systematic review of diagnosis and treatment. JAMA. 2013;310:1721-9 Everitt H, Wormald R, Henshaw K, et al. Viral conjunctivitis. In: Wormald R, Smeeth L, Henshaw K, eds. Evidence Based Ophthalmology. London: BMJ books, 2003</td>
</tr>
</tbody>
</table>

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**LAY SUMMARY**

Viral conjunctivitis is an infection of the eye in which one or both eyes become red and uncomfortable. The condition is not normally serious and in most cases clears up without treatment. It is highly infectious and care needs to be taken to prevent others from becoming infected, for example by not sharing towels.

In terms of treatment, antibiotics are ineffective against viruses and there is no effective anti-viral drug. Usual care involves the control of symptoms using cool compresses applied to the closed eyes, coupled with the use of lubricating eye drops and ointment.

In a small number of cases viral conjunctivitis can lead to the development of small opaque areas within the cornea (the clear window at the front of the eye), which can cause blurred vision. In such cases, and where there is severe inflammation, emergency referral to an ophthalmologist should be arranged.
### Conjunctivitis medicamentosa (also Dermatoconjunctivitis medicamentosa)

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Chemical irritation of ocular and/or adnexal tissues by a topically applied drug or cosmetic, or by environmental or occupational substances or Delayed hypersensitivity (cell-mediated) response to a topically applied drug, preservative or other excipient, or cosmetic Some drugs and preservatives may be both toxic and capable of inducing a delayed hypersensitivity response Whatever the mechanism, there may be a delay in onset of weeks or months following a symptom-free period</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predisposing factors</td>
<td>Topical ophthalmic medication (either prescribed or over the counter) Cosmetics applied to lids or lashes Related to dose and frequency If due to a delayed hypersensitivity response, takes at least two weeks to develop Some drugs and preservatives are more likely to produce the problem: • brimonidine • atropine • neomycin • benzalkonium chloride • phenylmercuric nitrate • lanolin (component of some eye ointments) Patients may use preserved topical medications for long periods of time, for example in glaucoma and in tear deficiency, but individual susceptibility to conjunctivitis medicamentosa varies widely</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Initial improvement in the original condition requiring treatment Then apparent deterioration despite proper compliance with regimen Irritation, ocular pain, stinging, burning, photophobia Ocular redness Lid swelling Blurred vision</td>
</tr>
<tr>
<td>Signs</td>
<td>Diffuse punctate staining of cornea and/or conjunctiva Chronic epithelial defects (due to toxic inhibition of epithelial healing) Tear film instability Sometimes: • corneal oedema • pseudodendrites • disciform stromal infiltrates</td>
</tr>
<tr>
<td>Differential diagnosis</td>
<td>Contact lens related staining or oedema Corneal erosion, abrasion or ulcer Endothelial dysfunction Rosacea Viral keratoconjunctivitis Dry eye, exposure keratitis Some topical medications (e.g. some prostaglandin analogues) cause hyperaemia as a known side effect</td>
</tr>
<tr>
<td>Management by Optometrist</td>
<td>Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere</td>
</tr>
<tr>
<td>Non pharmacological</td>
<td>Withdrawal of the offending medication or preservative Cold compress (symptomatic relief) Advise patient to avoid any future use of causative drug or preservative</td>
</tr>
</tbody>
</table>
**Pharmacological**

Non-prescribed (over the counter) medications:
- decide whether original condition still requires treatment
- prescribe unpreserved alternative if necessary

Prescribed medications:
- where unpreserved formulation of the same medication available, switch to that
- do not discontinue a medication when the consequences of interruption could be more serious than the conjunctivitis medicamentosa (e.g. glaucoma medications)
- refer back to original prescriber for consideration of alternative medication
- unpreserved tear supplements / ocular lubricants (for symptomatic relief)

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

- if severe, and in consultation with original prescriber, consider a short course of topical steroid, e.g. gutt. FML 0.1% qds for up to a week

(GRADE*: Level of evidence=low, Strength of recommendation=weak)

Antihistamines and mast cell stabilizers are not recommended for the treatment of conjunctivitis medicamentosa because they are ineffective in controlling inflammation in type IV hypersensitivity reactions

<table>
<thead>
<tr>
<th>Management Category</th>
<th>B2: Alleviation or palliation; normally no referral, but always inform and be advised by the original prescriber</th>
</tr>
</thead>
</table>

**Possible management by Ophthalmologist**

As above

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)

**Sources of evidence**


**LAY SUMMARY**

Conjunctivitis medicamentosa is a condition in which a drug applied to the eye as drops or ointment, or a cosmetic or some other substance reaching the eye surface, causes an irritative or allergic reaction. Some drugs are more likely than others to create this problem, including some anti-glaucoma agents and antibiotics. It can also be caused by preservatives in the medication.
Conjunctivitis medicamentosa (also Dermatoconjunctivitis medicamentosa)

The patient notices redness, stinging or burning and possibly eyelid swelling and/or blurred vision. Once recognised, the remedy involves withdrawing the offending medication and prescribing a substitute if necessary. If a preservative is the cause, it may be possible to obtain drops without preservative.
### Aetiology

Conjunctival pigmented lesions include a spectrum of benign, premalignant and malignant melanocytic conditions:

**Melanosis**
- Hypermelanosis (i.e. melanin overproduction by normal melanocytes)
  - racial melanosis
  - secondary melanosis (Addison’s disease; conjunctival lesions)
- Primary Acquired Melanosis (PAM), also known as Conjunctival Melanocytic Intraepithelial Neoplasia (C-MIN)
  - with or without 'atypia' (cellular structural abnormalities), graded according to cytomorphology, melanocytic density and spread to superficial layers of epithelium. Severe disease amounts to Melanoma In Situ (i.e. confined to epithelium)
- Congenital Melanocytosis
  - hyperpigmentation of episclera as a result of an overpopulation of melanocytes, also occurring in uvea and skin (i.e. Naevus of Ota). Predisposes to melanoma

**Naevus**
- the most common conjunctival pigmented lesion: 52% of ocular pigmented lesions
- congenital or acquired
- cluster of naevus cells in the conjunctival epithelium, usually extending to substantia propria. Cysts are often present

**Melanoma**
- rare malignant tumour arising from naevus, PAM or de novo: 3-5% of ocular malignancies, incidence 0.2-0.8 per million in Caucasian populations
- may arise in both sun-exposed and non-sun-exposed parts of the conjunctiva
- metastasises to regional lymph nodes and systemically, especially if involving caruncle and/or non-bulbar conjunctiva

Systemic disorders and drugs linked rarely with conjunctival pigmentation include:
- Addison’s disease (adrenal gland insufficiency)
- alcaptonuria (congenital enzyme deficiency)
- drugs (chlorpromazine, topical epinephrine, etc.)

### Predisposing factors

Epithelial melanosis is common in dark-skinned ethnicities.

PAM typically affects older white-skinned patients (rarely in dark-skinned)

Melanoma is more common in people with fair skin and blue eyes, extremely rare in dark-skinned races. Presentation peaks in mid-fifties

### Symptoms

Asymptomatic except for cosmetic concern

### Signs

**Ethnic melanosis**
Bilateral, asymmetrical, flat, intra-epithelial (moves freely over sclera), patchy, brown pigmentation, most prominent in palpebral aperture especially at limbus or where anterior ciliary arteries perforate the sclera, develops in early years (static by adulthood)

**C-MIN/PAM**
Unilateral, any part of conjunctiva (including tarsal or fornical), flat, intra-epithelial (moves freely over sclera), single or multiple, indistinct areas, light to dark brown, no cystic spaces, often extensive, can be stable or may change (enlarge, shrink, darken or lighten)

**Congenital Melanocytosis**
- **ocular**
  - Multifocal, slate-grey or blue grey, sub-epithelial (does not move freely over sclera)
- **dermal**
  - Mottled, blue to purple, discoloration of skin around the eye

**Naevus**
Solitary, sharply-demarcated, flat or slightly-elevated, intra-epithelial (moves freely over sclera). Confined to interpapillary zone (very rare in palpebral or fornical conjunctiva), most commonly adjacent to but not touching the limbus (less frequently at plica, caruncle, lid margin). NB: a pigmented lesion that straddles the peripheral cornea is highly suspicious and should be assumed to be a malignant melanoma. Presents in second or third decade when naevus becomes pigmented. Colour ranges from deep brown through pink to barely perceptible pigment. Often contains cystic spaces. Very rarely vascularised or inflamed

**Melanoma**
Nodular, well-vascularised mass with large conjunctival feeder vessels, fixed to underlying sclera (assess the degree of tethering, under topical anaesthesia). May be pigmented or non-pigmented (amelanotic), and nodular, diffuse or mixed. Clinicopathological features significantly associated with a poor prognosis are extrabulbar location, involvement of adjacent tissue structures, tumour thickness exceeding 2 mm and local tumour recurrence

| Differential diagnosis | Conjunctival intraepithelial squamous neoplasia (i.e. carcinoma in situ) can resemble an amelanotic melanoma
|                        | Blue naevus: a rare congenital deeply pigmented melanocytic lesion which can resemble PAM or melanoma |

**Management by Optometrist**
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

**Non pharmacological**
General advice on ocular UV protection
(GRADE*: Level of evidence=low, Strength of recommendation=strong)
# Conjunctival pigmented lesions

<table>
<thead>
<tr>
<th>Ethnic melanosis</th>
<th>Has no malignancy potential and requires no treatment <em>(GRADE</em>: Level of evidence=low, Strength of recommendation=strong)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PAM / C-MIN</strong></td>
<td>Sometimes has potential for malignancy (up to 13%) – refer for assessment, which requires biopsy to identify atypia <em>(GRADE</em>: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
<tr>
<td><strong>Congenital Ocular Melanocytosis</strong></td>
<td>Has potential for malignancy – refer. Is associated with malignant melanoma of the affected skin, orbit and uveal tract (fundoscopy with pupillary dilatation is required). Also associated with hyperpigmentation elsewhere in the eye including the trabeculum (regular monitoring for glaucoma required) <em>(GRADE</em>: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
<tr>
<td><strong>Naevus</strong></td>
<td>Generally requires no treatment, but very rarely progresses to a malignant melanoma. Advise patient to report any increase in size, elevation or colour. Review after 6 months and then every 12 months if lesion unaltered. Photo-document if possible <em>(GRADE</em>: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
<tr>
<td><strong>Melanoma</strong></td>
<td>Refer urgently (potentially sight- and life- threatening). Disseminates by local extension and by spread via lymphatic system (check preauricular and submandibular lymph nodes) <em>(GRADE</em>: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

**Pharmacological Management** None

**Management Category**

- **B1**: Routine referral to ophthalmologist
  - PAM /C-MIN
  - Naevus, especially if non-bulbar conjunctiva is involved
  - Congenital Ocular Melanocytosis

- **B2**: Alleviation/palliation: normally no referral to ophthalmologist
  - Mild ethnic melanosis

- **A3**: Urgent (within one week) referral to ophthalmologist
  - Melanoma

**Possible management by Ophthalmologist**

- PAM requires multiple biopsies to detect the histopathological characteristics that predict invasive melanoma. Tests for malignancy and excision where required
- Referral to regional specialist centre for biopsies and/or treatment
- Melanoma is usually treated by *en bloc* surgical excision with adjuvant therapy such as: mitomycin C, radiotherapy, topical chemotherapy. More than 50% develop local tumour recurrence with 20% requiring
Conjunctival pigmented lesions

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

**Sources of evidence**


**LAY SUMMARY**

The conjunctiva (the transparent skin over the white of the eye) sometimes develops brown discolouration. This is classified according to the cause:

- **Hypermelanos**: melanocytes (the cells of the body that produce the dark pigment melanin) go into overproduction. This may be a normal racial characteristic, or it may be caused by disease elsewhere in the body.
- **Primary Acquired Melanosis**: unusually large numbers of melanocytes develop. This is rare in dark-skinned races and tends to affect older white-skinned people.
- **Congenital Melanocytosis**: similar, except present from birth.

A Naevus, that is a brown spot on the conjunctiva, may be present from birth or may arise later. This is the commonest of all the conjunctival pigmented lesions. Usually it does not grow or spread.

Sometimes, a naevus changes into an Invasive Melanoma, also known as a Malignant Melanoma, which can spread to other parts of the body. This particularly affects people with fair complexions and is seen only very rarely in dark-skinned people.
There are also some uncommon generalised diseases that may produce discolouration of the conjunctiva. Some drugs may also cause a similar effect.

Depending on the nature of the pigmented lesion, optometrists may monitor the condition themselves, or refer to an ophthalmologist routinely or urgently. Mild ethnic melanosis does not need to be referred.

The ophthalmologist will carry out tests to identify which condition the patient has. Melanoma is usually treated with surgery and additional drug therapy. Careful follow-up is required.
# Conjunctival scarring

## Aetiology

All conjunctival scarring is, by definition, ‘cicatricial’; but the term ‘cicatrising conjunctivitis’ is generally reserved for scarring in which there is significant tissue shrinkage, usually with distortion of the fornices and/or the lids.

Many conditions can cause conjunctival scarring:

- **Severity** ranges from trivial to sight threatening.
- **Many potential causes:**
  - **Trauma**
    - surgery
    - thermal, radiation, mechanical, chemical
    - trichiasis
    - entropion (see Clinical Management Guideline)
  - **Exposure**
    - entropion (see Clinical Management Guideline)
    - lagophthalmos and other disturbance of lid function
  - **Autoimmune**
    - ocular cicatricial pemphigoid (OCP)
    - Stevens-Johnson syndrome (erythema multiforme major)
    - graft versus host disease
  - **Infection** (N.B. very few forms of infective conjunctivitis cause scarring)
    - trachoma (recurrent infection by *Chlamydia trachomatis* [serotypes A-C])
  - **Allergy**
    - vernal keratoconjunctivitis (see Clinical Management Guideline)
    - atopic keratoconjunctivitis (see Clinical Management Guideline)
  - **Ligneous conjunctivitis**
    - rare form of chronic conjunctivitis characterised by pseudomembranous lesions of ‘woody’ consistency

## Predisposing factors

OCP is primarily a disease of the elderly.

Stevens-Johnson syndrome usually occurs in previously healthy young adults:

- hypersensitivity reaction precipitated by many different antigens including
  - bacteria, viruses, fungi, drugs
- Trachoma: a disease of under-privilege and compromised hygiene
  - globally, the leading infectious cause of blindness

Atopic keratoconjunctivitis typically affects young atopic adults.

## Symptoms

Symptoms depend on severity and type of scarring:

Reduced tear components and compromised lid function both lead to dry eye:

- grittiness, burning, foreign body sensation
- blurred vision in severe cases

## Signs

Depends on aetiology
Conjunctival scarring

<table>
<thead>
<tr>
<th>Surgical and traumatic scarring</th>
<th>OCP produces sequence of conjunctival changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• focal, linear or diffuse scarring according to cause</td>
<td>• bilateral (often asymmetrical)</td>
</tr>
<tr>
<td>• diffuse hyperaemia, papillae</td>
<td>• diffuse hyperaemia, papillae</td>
</tr>
<tr>
<td>• bullae leading to ulceration and pseudomembrane formation</td>
<td>• bullae leading to ulceration and pseudomembrane formation</td>
</tr>
<tr>
<td>• subepithelial fibrosis and shrinkage; sometimes symblepharon</td>
<td>• subepithelial fibrosis and shrinkage; sometimes symblepharon</td>
</tr>
<tr>
<td>• secondary corneal changes</td>
<td>• secondary corneal changes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Stevens-Johnson syndrome produces sequence of conjunctival changes</th>
<th>Vernal keratoconjunctivitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• acute bilateral mucopurulent conjunctivitis</td>
<td>• tarsal sub-conjunctival fibrosis</td>
</tr>
<tr>
<td>• fibrosis and keratinisation follow acute phase</td>
<td>• pannus (especially at upper limbus)</td>
</tr>
<tr>
<td>• secondary corneal changes due to tear deficiency, exposure, keratinisation of the tarsal conjunctiva</td>
<td>• secondary corneal changes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vernal keratoconjunctivitis</th>
<th>Atopic keratoconjunctivitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• tarsal sub-conjunctival fibrosis</td>
<td>• tarsal sub-conjunctival fibrosis</td>
</tr>
<tr>
<td>• pannus (especially at upper limbus)</td>
<td>• conjunctival shrinkage</td>
</tr>
<tr>
<td>• forniceal shortening</td>
<td>• forniceal shortening</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Trachoma</th>
<th>Differential diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• follicles (upper tarsus)</td>
<td>A comprehensive ophthalmic and medical history should reveal the cause</td>
</tr>
<tr>
<td>• pannus (especially at upper limbus)</td>
<td>If no apparent cause, rule out early stages of OCP</td>
</tr>
<tr>
<td>• conjunctival inflammation leading to scarring and trichiasis</td>
<td></td>
</tr>
<tr>
<td>- Von Arlt's line (horizontal line of scarring parallel to lid margin)</td>
<td></td>
</tr>
<tr>
<td>• Herbert's pits (depressions at the upper limbus representing resolved limbal follicles)</td>
<td></td>
</tr>
<tr>
<td>• secondary corneal changes</td>
<td></td>
</tr>
</tbody>
</table>

**Management by Optometrist**

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

<table>
<thead>
<tr>
<th>Non pharmacological</th>
<th>Check for signs of dry eye</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• reduced tear meniscus</td>
</tr>
<tr>
<td></td>
<td>• low tear break up time</td>
</tr>
<tr>
<td></td>
<td>• fluorescein (drops or diagnostic strips) for cornea</td>
</tr>
</tbody>
</table>

**Check for signs of mechanical trauma to cornea due to:**

- trichiasis, keratinised tarsal conjunctiva

<table>
<thead>
<tr>
<th>Taping to reduce entropion (temporary measure)</th>
<th>Taping lids together at night in lagophthalmos</th>
</tr>
</thead>
<tbody>
<tr>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

**Therapeutic contact lens. All types of lens have been used. When the eye is relatively dry the first choice may be a scleral lens:**

- to prevent desiccation by maintaining a fluid layer beneath the
Conjunctival scarring

| Pharmacological | Ocular lubricants for tear deficiency/instability related symptoms (drops for use during the day, unmixed ointment for use at bedtime)  
*NB Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Clinical Management Guideline on Conjunctivitis Medicamentosa). They should be switched to unpreserved preparations*  
(GRADE*: Level of evidence=low, Strength of recommendation=weak) |
| Management Category | Mild scarring resulting from minor trauma or surgery:  
**B2**: alleviation / palliation: normally no referral  
Moderate to severe scarring:  
**B1**: initial management (including drugs) followed by routine referral  
Any conjunctival scarring of unknown aetiology should be referred  
**A3**: Urgent referral (within one week) if ocular autoimmune disease is suspected |
| Possible management by Ophthalmologist |  
• Topical and/or systemic immunosuppression if autoimmune aetiology  
• Conjunctival or buccal mucus membrane grafts, stem cell transplantation, amniotic membrane transplantation, lid surgery (but surgery in OCP can stimulate inflammation and produce further scarring) |

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see [http://gradeworkinggroup.org/toolbox/index.htm](http://gradeworkinggroup.org/toolbox/index.htm))

**Sources of evidence**


Sharma N, Thenarasing SA, Kaur M, Pushker N, Khanna N, Agarwal T, Vajpayee RB. Adjuvant Role of Amniotic Membrane Transplantation in
Conjunctival scarring


LAY SUMMARY

Many conditions can cause the conjunctiva, the thin transparent membrane covering the white of the eye and the underside of the eyelids, to become scarred. These include injury, infection, allergy and autoimmune diseases, in which the body’s immune system attacks its own cells or tissues. On a global scale, a major cause of conjunctival scarring and blindness is an infectious disease called trachoma, which is not common in the UK but affects many millions of people in North Africa and South Asia.

Scarring damages the conjunctiva and makes it less able to retain tears and protective mucus. Patients may have symptoms of dry eye, with grittiness, burning and, in severe cases, blurred vision. They may be helped by artificial tear drops, eyelid surgery and transplantation of amniotic membrane (innermost layer of the placenta) on to the eye surface.
### Aetiology

Loss of corneal epithelial tissue due to:
- sub-tarsal foreign body
- trauma (e.g. fingernail, twig, edge of paper, mascara brush)
- contact lens related trauma
- trichiasis (e.g. lash contact in entropion)

### Predisposing factors

Contact lens wear
- Epithelial Basement Membrane (EBM) dystrophy, in which epithelium is abnormal and easily traumatised
- Corneal exposure
  - dry eye
  - lagophthalmos
  - facial palsy
- Diabetes
- Neurotrophic keratitis

### Symptoms

Pain
- sudden onset
- ranges from mild foreign body sensation to severe pain; may be disproportionate to objective findings
- absence of pain should alert to possibility of neurotrophic keratitis
- Blepharospasm
- Photophobia
- Lacrimation
- Redness
- History of trauma

### Signs

Vary according to severity of trauma
- Lid oedema and erythema
- Conjunctival hyperaemia
- Corneal epithelial defect (stains with fluorescein)
- Corneal oedema beneath defect
- Visual loss (due to epithelial disruption and stromal oedema)
- Possible secondary anterior uveitis (anterior ciliary injection, cells, flare)

### Differential diagnosis

- Infectious keratitis (all forms)
- Recurrent corneal erosion
- Spontaneous epithelial breakdown in EBM Dystrophy (see Clinical Management Guideline on Recurrent Erosion Syndrome)
- Photokeratitis (see Clinical Management Guideline on Photokeratitis)

### Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

**Non pharmacological**
- Determine how the injury was caused. In particular rule out chemical injury and penetrating trauma
- Evaluate abrasion using fluorescein
  - size (use length of slit beam) and location
  - depth
  - edge quality
  - oedema beneath abrasion
  - confirm no corneal foreign body present

Corneal abrasion
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Date of search 26.03.19; Date of revision 04.07.19; Date of publication ab.cd.ef; Date for review 25.03.21
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If corneal foreign body present, see Clinical Management Guideline on Corneal Foreign Body
Evaluate anterior chamber reaction
Evert eyelids to confirm no foreign body present
If sub-tarsal foreign body present, see Clinical Management Guideline on Sub-Tarsal Foreign Body
Advise patient to return/seek further help if symptoms persist (potential for development of Recurrent Epithelial Erosion Syndrome (see CMG on Recurrent Epithelial Erosion Syndrome)
Advise on suitable eye protection

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

For large abrasions, consider therapeutic contact lens fitting

(GRADE*: Level of evidence=low, Strength of recommendation=weak)

Do not patch or pad eye

(GRADE*: Level of evidence=high, Strength of recommendation=strong)

Pharmacological
Topical anaesthetic (e.g. gutt. proxymetacaine 0.5% or gutt. oxybuprocaine 0.4%) if necessary to aid examination
Systemic analgesia for first 24h (e.g. ibuprofen, or paracetamol if contraindicated)

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Ocular lubricants for symptomatic relief (drops as needed for use during the day, unmedicated ointment for use at bedtime)

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

If there is a possibility of infection, prescribe a broad spectrum topical antibiotic e.g. gutt. chloramphenicol 0.5% qds for five days (NB risk of infection following mild trauma is low)

(GRADE*: Level of evidence=low, Strength of recommendation=weak)

In the case of contact lens wearers, antibiotic prophylaxis should be with a drug effective against Gram -ve organisms, e.g. a quinolone such as levofloxacin or moxifloxacin, or an aminoglycoside such as gentamicin.
Contact lenses should not be worn during the treatment period

(GRADE*: Level of evidence = low, Strength of recommendation = strong).

Topical NSAID for its analgesic and anti-inflammatory properties, e.g. gutt. diclofenac 0.1% up to four times daily for 1-3 days
RCTs do not provide strong evidence to support their use

(GRADE*: Level of evidence=moderate, Strength of recommendation=weak)

For large abrasions or in associated iritis, consider cycloplegia to prevent pupil spasm, (e.g. gutt. cyclopentolate 1% twice daily until healed)

(GRADE*: Level of evidence=low, Strength of recommendation=weak)

Management Category
B3: management to resolution
A2: if abrasion deep and/or contaminated with foreign material, or apparently infected, refer as emergency (same day) to Ophthalmologist
Corneal abrasion

Possible management by Ophthalmologist

- Assess for secondary infection
- Debridement if indicated
- Therapeutic contact lens fitting
- Plain X-ray or CT scan to exclude retained foreign body

Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence

Corneal Abrasions. BMJ Best Evidence 2018
https://bestpractice.bmj.com/topics/en-gb/500


LAY SUMMARY

Abrasions of the cornea (the clear window of the eye) are common, being usually caused by a minor accidental injury, for example by a finger, mascara brush or contact lens, or by a speck of foreign matter under the upper eyelid. There are also medical conditions that make abrasions more likely, for example a condition, known as a dystrophy, in which the surface tissue of the cornea (the epithelium) is more delicate than usual; also when the cornea is exposed by failure of the normal blink reflex, or when its sensitivity to touch is reduced by damage to its nerves, as in diabetes or following shingles of the eye. Corneal abrasion can be very painful as the cornea is one of the most sensitive areas of the body.

The clinician will assess the area involved and prescribe treatment accordingly. The damage to the surface can be seen more easily if fluorescein, an orange dye, is instilled into the eye. Anti-inflammatory or antibiotic eye drops are often recommended, depending on the type and size of abrasion. Dilating eye drops are sometimes given to relieve discomfort. There is little supporting evidence for the use of these drugs.

Corneal abrasions usually heal quickly and completely but if the injury is deeper, or contaminated by foreign material, or possibly infected, referral to an ophthalmologist is recommended.
# Corneal (or other superficial ocular) foreign body

## Aetiology
Patient often gives history of foreign body entering eye
- wind blown
- high velocity (hammering, grinding)
- DIY and gardening

## Predisposing factors
Lack of suitable eye protection

## Symptoms
Typically unilateral
- Irritation/foreign body sensation/pain
- Lacrimation
- Blurred vision
- Red eye

## Signs
- Foreign body adherent to ocular surface
- Linear corneal scratches
- Corneal rust ring from ferrous foreign body
- Surrounding ring of oedema and infiltrate if longstanding
- Subconjunctival haemorrhage may be present

## Differential diagnosis
History is important
- high velocity particles – risk of globe penetration
- metallic (ferrous) – rust ring (haemosiderosis)
- vegetative – risk of fungal infection
- Recurrent erosion syndrome

## Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

### Non pharmacological
Rule out multiple particles – cornea, conjunctiva (bulbar, fornix, palpebral): double evert lids (e.g. using a Desmarre’s retractor)
- loose foreign body can be irrigated away with normal saline
- foreign body on conjunctiva can be removed with a sterile cotton bud
- assess depth of corneal foreign body (slit lamp optical section)
- carry out Seidel test to check for corneal perforation
- corneal foreign body may require removal with a hypodermic needle or other disposable instrument. To reduce the risk of corneal penetration, ensure that the needle approaches the cornea tangentially
- after removal, assess size of remaining epithelial defect so that healing can be monitored

Check:
- VA before and after FB removal
- globe and adnexae for signs of penetration
- where there is any suspicion of a penetrating injury, carry out dilated fundus examination
- AC for flare or cells
- pupil responses

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

For those specifically trained, use of burr or other instrument (e.g. Alger Brush) to remove rust ring
- if non-disposable instruments are used they must come from a
## Corneal (or other superficial ocular) foreign body

<table>
<thead>
<tr>
<th>Sterile pack</th>
</tr>
</thead>
<tbody>
<tr>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=weak)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Do not pad or patch eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>(GRADE*: Level of evidence=high, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Advise patient to return/seek further help if symptoms persist</th>
</tr>
</thead>
<tbody>
<tr>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

### Management Category

<table>
<thead>
<tr>
<th><strong>B3</strong>: superficial FB: management to resolution, normally no referral</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A2</strong>: penetration into stroma, or presence of rust ring, may result in scarring and potential visual loss, therefore refer to ophthalmologist as emergency (same day); (but note exception for optometrists specifically trained in rust ring removal)</td>
</tr>
</tbody>
</table>

### Pharmacological

<table>
<thead>
<tr>
<th>Remove foreign body under topical anaesthesia (gutt. proxymetacaine 0.5% or gutt. oxybuprocaine 0.4%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consider use of ointment (unmedicated or medicated) following removal (as ocular lubrication)</td>
</tr>
<tr>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

If there is a likelihood of infection, consider topical antibiotic prophylaxis (e.g. gutt. chloramphenicol 0.5% qds for 5 days) For large epithelial defects, cycloplegia to prevent pupil spasm (e.g. gutt cyclopentolate 1% twice daily until healed) (GRADE*: Level of evidence=low, Strength of recommendation=weak)

In the case of contact lens wearers, antibiotic prophylaxis should be with a drug effective against Gram -ve organisms, e.g. a quinolone such as levofloxacin or moxifloxacin, or an aminoglycoside such as gentamicin. Contact lenses should not be worn during the treatment period (GRADE*: Level of evidence = low, Strength of recommendation = strong)

Systemic analgesia (e.g. ibuprofen, or paracetamol if contraindicated) if necessary (GRADE*: Level of evidence=low, Strength of recommendation=strong)

Topical NSAID for its analgesic and anti-inflammatory properties, e.g. gutt. diclofenac 0.1% up to four times daily for 1-3 days RCTs do not provide strong evidence to support their use (GRADE*: Level of evidence=moderate, Strength of recommendation=weak)

### Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*
Corneal (or other superficial ocular) foreign body

Sources of evidence

Corneal Abrasions. BMJ Best Evidence 2018
https://bestpractice.bmj.com/topics/en-gb/500


LAY SUMMARY

Small foreign bodies commonly enter the eye. Usually these are blinked away but sometimes they adhere to the surface of the cornea (the clear window of the eye). This is more likely if they enter at high speed, for example when blown in by the wind or when they result from hammering, grinding, other DIY and gardening activities.

If a foreign body becomes attached to the cornea it can be removed with a delicate instrument after the surface of the eye has been numbed by an anaesthetic drop. If the foreign body is iron-based, it may have quickly rusted on the eye surface, in which case the rust will need to be removed also. If there is a likelihood of infection, antibiotic drops and/or ointment may be prescribed. Patients will be referred to the ophthalmologist only if the foreign body has penetrated below the surface or is likely to cause corneal scarring.
**Fuchs Endothelial Corneal Dystrophy (FECD)**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Fuchs Endothelial Corneal Dystrophy (FECD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aetiology</strong></td>
<td>The most common posterior corneal dystrophy, characterised by a slowly progressive dysfunction of the corneal endothelium that eventually results in corneal oedema and reduced vision; resultant stromal and epithelial oedema leads to epithelial bullae. Studies of familial FECD cases show that the disease has an autosomal dominant inheritance pattern. However, the majority of patients with FECD have sporadic disease, without a familial history. The ICD3 classification categorises FECD patients as those with: 1) early-onset FECD, 2) identified genetic loci, and 3) disease without known inheritance.</td>
</tr>
<tr>
<td><strong>Predisposing factors</strong></td>
<td>Most cases begin in the fourth decade or later. Female: male predominance = 2.5:1 to 3:1. A rare early-onset variant begins in the first decade of life.</td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td>None in the early stages. Symptoms rare under age 50. Glare and blurred vision especially on waking (symptoms accentuated by overnight corneal oedema). Diurnal changes in refraction (relatively myopic on waking). Sharp pain (when epithelial bullae rupture).</td>
</tr>
<tr>
<td><strong>Differential diagnosis</strong></td>
<td>Pseudophakic or aphakic bullous keratopathy. Posterior polymorphous dystrophy. Cornea guttata (often listed as a dystrophy, but not necessarily progressive). Corneal hydrops (in keratoconus). Contact lens overwear.</td>
</tr>
</tbody>
</table>

**Management by Optometrist**

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.

**Non pharmacological**

- Photography and imaging, including specular microscopy (if available)
- Measurement of central corneal thickness (CCT) for monitoring progression of disease (GRADE*: Level of evidence=low, Strength of recommendation=strong)
- Where there is pain, a therapeutic contact lens may protect exposed corneal nerves and reduce friction between lid margins and bullae on blinking.
## Fuchs Endothelial Corneal Dystrophy

### Pharmacological

Table: Pharmacological Management

<table>
<thead>
<tr>
<th>Management Category</th>
<th>B1: initial management, possible routine referral (modified statement)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oc. sodium chloride 5% or gutt. sodium chloride 5%</td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
<tr>
<td>Oc. sodium chloride 5% or gutt. sodium chloride 5% (in severe cases with persistent oedema) applied in the mornings and as necessary. The dosage will need to be varied as usefulness or otherwise in controlling the patient’s symptoms becomes apparent</td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

### Management

**Management Category**

Possible management by Ophthalmologist

- Confirmation of diagnosis
- Photography and imaging, including specular microscopy
- Measurement of CCT for monitoring progression of disease
- Repeat assessment to establish progression
- When condition no longer responsive to management of symptoms, may offer surgery:
  - posterior lamellar transplantation (currently the preferred treatment for irreversible corneal endothelial decompensation);
  - techniques include:
    - Descemet’s Stripping Automated Endothelial Keratoplasty, DSAEK
    - Descemet’s Stripping Endothelial Keratoplasty, DSEK
    - Descemet’s Membrane Endothelial Keratoplasty, DMEK
  - penetrating keratoplasty
  - either of the above procedures may be combined with cataract surgery if this is clinically indicated

Endothelial keratoplasty procedures have largely superseded PK for FECD, although the superiority of these techniques has not been demonstrated using RCTs. Prospective national registry data from Australia and the UK shows higher transplant failure rates than are seen following PK. These higher failure rates of EK compared with PK may be acceptable given the established benefits of the procedure, including lower refractive error, structural globe integrity, and faster visual recovery

### Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)

Sources of evidence

- Knezović I, Dekaris I, Gabrić N, Cerovski J, Barisić A, Bosnar D, Rastegorac P,
### Fuchs Endothelial Corneal Dystrophy

<table>
<thead>
<tr>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parać A. Therapeutic efficacy of 5% NaCl hypertonic solution in patients with bullous keratopathy. Coll Antropol. 2006;30:405-8</td>
</tr>
</tbody>
</table>

### Note on Corneal Dystrophies

Corneal dystrophies are progressive, usually bilateral, mostly inherited, alterations in corneal transparency that develop without inflammation. In the past, the nomenclature of the many dystrophies has often caused confusion. A newer classification (the IC3D Classification) has been proposed which integrates up-to-date information on phenotypic description, histopathological examination and genetic analysis: [http://eyepath.org.uk/wp-content/uploads/2016/02/IC3D_Classification_of_Corneal_Dystrophies_Edition.2.pdf](http://eyepath.org.uk/wp-content/uploads/2016/02/IC3D_Classification_of_Corneal_Dystrophies_Edition.2.pdf)

In everyday clinical practice, the most commonly encountered corneal dystrophies are Epithelial Basement Membrane Dystrophy (Map-Dot-Fingerprint Dystrophy, Cogan’s Dystrophy), which affects the epithelium and predisposes to Recurrent Corneal Epithelial Erosion (see the Clinical Management Guideline of that name) and Fuchs Endothelial Corneal Dystrophy, the subject of this Clinical Management Guideline.

### LAY SUMMARY

Fuchs Endothelial Corneal Dystrophy

Version 13, Page 3 of 4

Date of search 22.11.18; Date of revision 29.03.19; Date of publication ab.cd.ef; Date for review 21.11.20

© College of Optometrists
Fuchs Endothelial Corneal Dystrophy

A corneal dystrophy is a condition in which the cornea (the clear window of the eye) loses its normal transparency. It is usually inherited, affects both eyes and is progressive. There is no inflammation or infection. This particular corneal dystrophy was first described by an Austrian ophthalmologist, Ernst Fuchs, in 1910, and it has borne his name ever since. In this condition the cells lining the back of the cornea (the endothelial cells), which normally pump water out of the cornea, keeping it clear, no longer work properly. As a result the cornea becomes water-logged and cloudy and the vision becomes hazy. The condition is painless at first, but if, at a later stage, blisters form on the surface of the cornea and then burst, sharp pain may result. No treatment is available to reverse this condition but if the vision is greatly affected or the eye is painful, the ophthalmologist may recommend a corneal transplant procedure. This has a good chance of improving the vision and making the eye comfortable.
### Aetiology
Conventionally believed to be due to rupture of Descemet’s membrane
Acute leakage of aqueous into corneal stroma and epithelium
The estimated UK annual incidence of acute corneal hydrops in keratoconus is 1.4 per 1000
Most cases occur in second or third decade with men affected 2-3 times more than women

### Predisposing factors
Keratoconus, keratoglobus, pellucid marginal degeneration or other primary corneal ectasia; high risk in advanced keratoconus
Vernal keratoconjunctivitis
Asthma
Eye rubbing may be a risk factor

### Symptoms
Sudden reduction in visual acuity
Discomfort
Photophobia
Watering
Contact lens intolerance

### Signs
Gross stromal oedema with or without epithelial oedema
• usually over a clearly demarcated area
  – Descemet’s membrane rupture may be visible
  – periphery usually spared, except in pellucid marginal degeneration

### Differential diagnosis
Other causes of corneal oedema including Fuchs dystrophy and infective keratitis

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

#### Non pharmacological
**Acute hydrops**
- cease contact lens wear
- avoid bandage contact lens (hypoxia may induce corneal vascularisation)
- initially, review weekly for appearance of vascularisation or other complication
  – majority of cases resolve over 2-4 months, sometimes with stromal scarring
  – if corneal vascularisation appears, refer urgently to ophthalmologist

**After resolution**
- reassess corneal topography (often less steep after hydrops)
- will probably still need contact lens for optimum acuity; lens fit will need to be reviewed as corneal profile may have changed

**Pharmacological**
Topical lubricants for symptomatic relief

**Cycloplegia for symptomatic relief, e.g. gutt. cyclopentolate 1% twice daily for at least a week**

### Notes
GRADE*: Level of evidence=low, Strength of recommendation=strong
GRADE*: Level of evidence=low, Strength of recommendation=strong
GRADE*: Level of evidence=low, Strength of recommendation=weak
Corneal hydrops

Systemic analgesia (e.g. ibuprofen, or paracetamol if contraindicated) if necessary

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Consider prophylactic topical antibiotic (e.g. gutt. chloramphenicol 0.5% qds) if epithelial surface acutely disturbed by oedema and if secondary infection seems likely to occur

(GRADE*: Level of evidence=low, Strength of recommendation=weak)

Referral (Category)

B2: Alleviation/palliation: normally no referral
A3: first aid measures and urgent referral (if vascularisation present)

Possible management by Ophthalmologist

Treat pain, prophylactic antibiotic if indicated, topical steroid if corneal vessels proliferate, penetrating keratoplasty if scarring reduces acuity following resolution
Possible intracameral gas injection

Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence


This is a rare occurrence seen occasionally in people in whom the cornea (the clear window of the eye) is thinned and distorted, for example in the condition known as keratoconus. Usually for no apparent reason, the back membrane of the cornea splits, allowing fluid from within the eye to flood into the cornea which then loses its clarity. The vision may be badly affected.

The condition usually improves by itself over a period of 2-4 months. If there is any complication in the recovery period, for example if new blood vessels appear in the cornea, patients are quickly referred to the ophthalmologist for specialist treatment.

These patients are usually wearing contact lenses to enhance their vision because glasses do not help very much. The lenses may need to be refitted when the condition improves, because of a change in shape of the eye.
## Aetiology

Corneal transplant rejection is the most common cause of transplant failure in the post-operative period. 30% of transplanted corneas experience at least one episode of immune rejection, and a proportion of these lead to eventual transplant failure. The incidence of graft rejection depends on the presence of risk characteristics, e.g. corneal neovascularization.

Corneal transplant rejection may be reversible or irreversible and can affect both full thickness and lamellar transplants, although rejection may be less common following lamellar procedures.

## Predisposing factors

- host corneal stromal vascularisation
  - for example, risk doubled if all four quadrants involved
- young recipient
- donor/recipient gender mismatch
  - male donor to female recipient
- large diameter or eccentric transplant
- loose or exposed sutures
- recent decrease in steroid therapy
- previous rejection
- previous transplant in the same eye
- iris adhesion to transplant/host interface
- time since operation: most rejections occur with one year of surgery, but can occur at any time
- *Herpes simplex*, bacterial or other infection
- inflammatory disease (e.g. anterior uveitis)
- glaucoma
- subsequent intra-ocular surgery (e.g. cataract surgery, vitrectomy)
- tear deficiency
- trauma (chemical, mechanical)

## Symptoms

- photophobia
- redness (may be perilimbal)
- epiphora
- blurred vision
- discomfort or pain

## Signs

Following full-thickness corneal transplantation (Penetrating Keratoplasty, PK), rejection may involve any cellular layer of the cornea (epithelium, stroma or endothelium). Of these, endothelial rejection is potentially the most serious as it threatens the viability of the transplant. Endothelial rejection is also of concern following posterior lamellar transplantation (Descemet’s Stripping Automated Endothelial Keratoplasty, DSAEK, and Descemet’s Membrane Endothelial Keratoplasty, DMEK), although it does not occur following anterior lamellar transplantation (ALK).

Some or all of the following signs may be present:
- sub-epithelial opacities similar in appearance to adenovirus keratitis (Krachmer spots)
- anterior ciliary injection (perilimbal hyperaemia)
### Differential diagnosis
- transplant oedema (may be regional)
- rejection line
  - endothelial (Khodadoust line) or epithelial
- keratic precipitates (KP) on transplant endothelium
- anterior chamber flare and cells
- raised IOP

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

<table>
<thead>
<tr>
<th>Category</th>
<th>A1: emergency (same day) referral; no intervention. Telephone on-call ophthalmologist</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non pharmacological</td>
<td>None</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>None</td>
</tr>
</tbody>
</table>

### Possible management by Ophthalmologist
- possible admission to hospital
- intensive topical steroid therapy
- possible addition of systemic steroid
- possible immunosuppressant therapy e.g. ciclosporin, tacrolimus
- topical antivirals where there is a history of *Herpes simplex* infection

### Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

**Sources of evidence**


Corneal Transplant Rejection


LAY SUMMARY

After a patient has had a corneal transplant, in which tissue from a donor eye is placed or sewn into the eye, the body’s immune system can recognise the tissue as foreign and may start a reaction against it. This reaction, known as corneal transplant rejection, can usually be controlled if it is discovered early enough.

A patient with a corneal transplant rejection may experience discomfort or pain in the eye, redness, blurred vision and watering.

The seriousness of such a rejection depends on the type of transplant that was carried out. If it was a full-thickness transplant, rejection is likely to involve the deepest layer of the donor cornea, known as the endothelial layer. As a functioning endothelium is essential for the cornea to remain transparent, a rejection of this layer must be treated quickly and thoroughly.

The optometrist who diagnoses a corneal transplant rejection will refer the patient to the ophthalmologist as an emergency. This will allow treatment with the appropriate steroid and other drugs to begin as soon as possible.
Aetiology

Bacterial infection of lacrimal sac
Usually secondary to blockage of nasolacrimal duct
Commonest in infants and post-menopausal women
Relatively rare in older children
Infection may be due to Gram positive or Gram-negative organisms: *Staphylococcus aureus* and *Streptococcus pneumoniae* are the most common isolates amongst Gram-positive bacteria and *Haemophilus influenzae, Serratia marcescens* and *Pseudomonas aeruginosa* amongst Gram-negative bacteria

Predisposing factors

Maxillary sinusitis
Trauma to adjacent tissues
Nasal or sinus surgery
Congenital obstruction of nasolacrimal duct (see Clinical Management Guideline on Nasolacrimal Duct Obstruction)

Symptoms

Sudden onset
Pain
Tender swelling over lacrimal sac (anatomically located just below the medial palpebral ligament)
Epiphora
Fever (raised temperature)

Signs

Red, tender swelling centred over lacrimal sac and extending around the orbit
Purulent discharge expressible from one or both puncta when pressure is applied over the lacrimal sac (NB likely to be painful for patient)
Sac may discharge on to skin surface
(NB important to distinguish between acute dacryocystitis, in which sac is full of pus, and mucocoele in which sac is filled with mucoid material in the absence of infection)
Frequently, patients may present with conjunctivitis and preseptal cellulitis. Rarely, the infection extends behind the septum, causing orbital cellulitis

Differential diagnosis

Facial cellulitis, preseptal cellulitis, orbital cellulitis (check ocular motility and look for proptosis) (Refer to Clinical Management Guideline on Cellulitis [preseptal and orbital])
Acute frontal sinusitis (inflammation involves the upper eyelid)
Infection following superficial trauma/abrasion of skin
(See also Clinical Management Guideline on Dacryocystitis (chronic))

Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

Non pharmacological

Do not attempt to probe the lacrimal system during acute infection (risk of spreading infection)
(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Pharmacological

Topical antibiotic to prevent bacterial conjunctivitis: e.g. chloramphenicol drops and/or ointment for not less than 5 days
For mild and non-febrile cases, consider prescribing systemic antibiotic, e.g. co-amoxiclav or, where there is a penicillin allergy, erythromycin
(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Management Category

A2 (modified, as condition not sight-threatening): for severe cases and in all children, give first aid measures and refer as emergency (same day) to ophthalmologist or A&E Department. Cases are severe if patient has pyrexia and/or is systemically unwell or if an abscess has
**Dacryocystitis (acute)**

**Possible management by Ophthalmologist**

- Incision and drainage where appropriate
- Systemic (including parenteral) antibiotics
- Follow-up may include investigation and surgical intervention for nasolacrimal duct obstruction

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

*Sources of evidence*


**LAY SUMMARY**

Dacryocystitis means inflammation of the tear sac, the small chamber in which the tear fluid collects as it drains from the eye surface, which is beneath the skin alongside the inner corner of the eye. It is commonest in infants and middle-aged women and is usually caused by an infection by commonly occurring bacteria. It starts suddenly with pain and tenderness over the tear sac and the patient may quickly develop a raised temperature. The infection may also cause conjunctivitis (infection of the transparent membrane over the white of the eye) and cellulitis (infection of the soft tissues surrounding the eye). Sometimes the sac bursts, releasing pus on to the skin surface.

It is important to try to distinguish between this condition and a serious infection of the eye socket (orbital cellulitis) itself, especially in children, who should be referred to hospital the same day for emergency treatment. Treatment includes antibiotics, which may have to be given via a needle into a vein, and surgery to encourage pus from the infection to drain away.
# Dacryocystitis (chronic)

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Lacrimal sac develops a mucocoele:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• filled with mucoid material</td>
</tr>
<tr>
<td></td>
<td>• can sometimes be expressed by applying pressure over the sac</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Predisposing factors</th>
<th>History of recurrent or chronic unilateral conjunctivitis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Previous acute dacryocystitis</td>
</tr>
<tr>
<td></td>
<td>Chronic nasolacrimal duct obstruction</td>
</tr>
<tr>
<td></td>
<td>Facial fracture</td>
</tr>
<tr>
<td></td>
<td>Foreign bodies (e.g. punctal or canalicular plugs)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>One or more of the following:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• recurrent episodes of epiphora, plus swelling, tenderness and redness at medial canthus</td>
</tr>
<tr>
<td></td>
<td>• persistent redness at medial canthus</td>
</tr>
<tr>
<td></td>
<td>• persistent painless swelling at or below the medial canthus</td>
</tr>
<tr>
<td></td>
<td>• chronic epiphora</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs</th>
<th>Recurrent episodes similar to, but less severe than, acute dacryocystitis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Swelling at or below medial canthus</td>
</tr>
<tr>
<td></td>
<td>May be possible to express mucoid (opalascent) discharge</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differential diagnosis</th>
<th>Canaliculitis, sinusitis, sebaceous cyst, preseptal cellulitis, tumour or granulomatous lesion causing nasolacrimal obstruction (blood in tears may suggest this)</th>
</tr>
</thead>
</table>

## Management by Optometrist

**Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere**

### Non pharmacological

In adults, it has been proposed that patients with lacrimal sac swelling and suspicion of obstruction of the lacrimal drainage system should be treated conservatively, reserving surgery for cases that do not respond. For symptomatic relief, advise traditional remedies such as hot compresses and massage

*(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

### Pharmacological

If infection suspected, give topical antibiotic (e.g. chloramphenicol drops or ointment) for not less than five days; also as a prophylactic measure while awaiting surgery

*(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

### Management Category

- **B2:** alleviation/palliation (normally no referral)
- **B1:** if symptoms recurrent and persistent, refer routinely

### Possible management by Ophthalmologist

Arrange dacryocystogram (DCG: X-ray with contrast medium)

Possible surgery: dacryocystorhinostomy (DCR)

### Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*

**Sources of evidence**

from the tear sac to the inside of the nose. Finger pressure over the sac sometimes causes white mucus to appear at the openings of the tear passages at the inner corners of the eyelids; this may help in reaching a diagnosis. Patients usually complain of swelling and sometimes tenderness over the tear sac, plus watering of the eye.

If the condition results in repeated episodes of acute infection, antibiotics are given, both as eye drops and as medicines to be taken by mouth. In less acute cases, hot compresses and massage over the tear sac may relieve the patient’s symptoms. A special X-ray known as a dacrycystogram or DCG may help to show exactly where the tear duct blockage is, and this will help the eye surgeon to decide on whether surgery is necessary, and if so, of what kind. In a commonly performed operation known as a dacryocystorhinostomy or DCR, a new passage is created from the tear sac into the inner wall of the nose, so that the tears can drain directly without having to pass down the tear duct.
### Aetiology
Outward rotation of the eyelid margin (usually lower). Occurs in approx. 4% of the population over 50 (bilateral in 70%). Various causes:
- Involutional (age-related degeneration)
  - Most common
  - Horizontal lid laxity
  - Weakness of pretarsal part of orbicularis oculi muscle
  - Weakness of medial and lateral canthal tendons
- Cicatricial: scarring +/- contracture of skin and underlying tissues
  - Trauma
  - Burns
  - Skin tumours
  - Actinic skin changes due to prolonged sun exposure
- Paralytic
  - (refer to Clinical Management Guideline on Facial Palsy)
- Mechanical
  - Tumour at or near the lid margin
  - Lid swelling due to inflammation from infection or allergy
- Congenital
  - Rare bilateral condition

<table>
<thead>
<tr>
<th>Predisposing factors</th>
<th>Lid laxity increases with age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Sore, red, watery eye</td>
</tr>
<tr>
<td></td>
<td>Symptoms variable depending on severity</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs</th>
<th>Inferior lid margin not in contact with globe:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Region involved may be punctal, medial, lateral, or tarsal (complete)</td>
</tr>
<tr>
<td></td>
<td>Involutional ectropion typically begins medially; central lid margin and lateral lid may become involved later</td>
</tr>
<tr>
<td></td>
<td>Keratinisation of exposed tarsal conjunctiva</td>
</tr>
<tr>
<td></td>
<td>Lower punctum not in contact with tear meniscus:</td>
</tr>
<tr>
<td></td>
<td>If punctum is spontaneously visible at slit lamp, ectropion is present</td>
</tr>
<tr>
<td></td>
<td>Conjunctival hyperaemia</td>
</tr>
<tr>
<td></td>
<td>Exposure keratopathy</td>
</tr>
<tr>
<td></td>
<td>Epiphora</td>
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<tr>
<td></td>
<td>Mucus discharge</td>
</tr>
<tr>
<td></td>
<td>Distraction test</td>
</tr>
<tr>
<td></td>
<td>If lower lid can be pulled &gt;6mm from globe, it is lax</td>
</tr>
<tr>
<td></td>
<td>Positive test indicates canthal tendon laxity</td>
</tr>
<tr>
<td></td>
<td>Snap-back test</td>
</tr>
<tr>
<td></td>
<td>With finger, pull lower lid down towards inferior orbital margin</td>
</tr>
<tr>
<td></td>
<td>Release: lid should snap back</td>
</tr>
<tr>
<td></td>
<td>Positive test indicates poor orbicularis tone</td>
</tr>
</tbody>
</table>

### Differential diagnosis
Ectropion is a physical sign, rather than a disease entity.

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.

<table>
<thead>
<tr>
<th>Non pharmacological</th>
<th>Mild cases require no treatment:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Advise that lid rubbing may increase lid laxity</td>
</tr>
</tbody>
</table>
Taping the lids closed at night when there is a risk of corneal exposure
(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Therapeutic contact lens where constant protection from corneal exposure is indicated
(GRADE*: Level of evidence=low, Strength of recommendation=weak)

**Pharmacological**

Ocular lubricants for tear deficiency/instability related symptoms (drops for use during the day, unmedicated ointment for use at bedtime)

*NB Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Clinical Management Guideline on Conjunctivitis Medicamentosa). They should be switched to unpreserved preparations

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

**Management Category**

**Mild asymptomatic involutional cases**


**More severe cases possibly requiring surgery**

B1: Initial management followed by routine referral

**Possible management by Ophthalmologist**

Surgery:
- a variety of surgical procedures (choice determined by nature, position and degree of ectropion)
- tarsorrhaphy sometimes necessary

Surgery is indicated for:
- ocular surface exposure (increased risk of microbial keratitis)
- chronic epiphora or ocular irritation
- recurrent bacterial conjunctivitis
- poor cosmesis

There are no available data from randomised trials to provide evidence for the most effective intervention for the correction of involutional ectropion

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence


**LAY SUMMARY**
Ectropion is a condition in which the eyelid (usually the lower eyelid) becomes slack and is no longer in contact with the eyeball. The most common cause is loss of elasticity and muscle tone of the eyelids which happens as part of the ageing process. The affected eye becomes sore, red and watery. Patients may be helped by artificial tears and unmedicated ointments. If the eye does not close fully at night, it may need to be taped shut. Sometimes a bandage contact lens is fitted to protect the eye surface from drying. If these measures do not help, one of a number of possible surgical operations, usually carried out under local anaesthetic, may solve the problem.
Endophthalmitis (post-operative) (Exogenous endophthalmitis)

### Aetiology

Post-operative endophthalmitis is a rare but severe sight-threatening complication of ocular surgery e.g. cataract, corneal, glaucoma, retinal, and of intravitreal injections, e.g. anti-VEGF treatment.

Occurs most commonly as a complication of cataract surgery. Pooled estimates of incidence range from 1.09-2.65 per 1,000 cataract operations.

A systematic review has demonstrated the value of intracameral cefuroxime with or without topical levofloxacin in reducing the incidence of post-operative endophthalmitis.

Bleb-associated endophthalmitis has a reported incidence of 2.1% at an average 18 months following glaucoma drainage surgery.

Pooled estimate of endophthalmitis following anti-VEGF treatment is 3 per 10,000 injections.

Organisms (examples only, in descending order of frequency):
- *Staphylococcus* sp. (50.5% of culture-positive cases)
- *Streptococcus* sp. (12.1%)
- Gram negative sp. (10.3%)
- fungi (4.6%)

Onset may be acute (in first week) or chronic (in first month). 80% of cases present within 6 weeks of surgery.

Post-operative endophthalmitis may also be non-infective (retention of foreign material, e.g. cotton fibres, or caused by toxic substances, e.g. component of unsuitable irrigating fluid).

Endophthalmitis is associated with significant visual morbidity (approx. 40% <6/60 after treatment).

### Predisposing factors

**Surgical**
- increased operative time
- posterior capsular rupture
- wound leakage

**Sources of contamination:**
- patient’s own bacterial flora (skin, lids, conjunctiva, lacrimal apparatus)
- contaminated instruments, solutions, drapes, dressings, gloves
- (in corneal transplants) donor cornea

**Patient factors:**
- diabetes, immunosuppression, HIV infection

### Symptoms

**Acute presentation:**
- visual loss
- pain
- redness
- photophobia

Chronic presentation: similar, usually milder, delayed

### Signs

**Acute presentation:**
- lid oedema
- conjunctival chemosis and hyperaemia
- corneal haze
- cells and flare in AC; fibrinous exudate and/or hypopyon if severe
# Endophthalmitis (post-operative) (Exogenous endophthalmitis)

<table>
<thead>
<tr>
<th>Sign</th>
<th>Symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>pupil light reflex</td>
<td>may be sluggish or absent</td>
</tr>
<tr>
<td>IOP</td>
<td>can be normal, low or raised</td>
</tr>
<tr>
<td>vitritis (inflammation of the vitreous)</td>
<td>may eliminate red reflex and preclude view of fundus</td>
</tr>
</tbody>
</table>

**Chronic presentation:** similar, usually milder, delayed

**Differential diagnosis**
- Post-operative inflammation without infection
- Other causes of acute red eye, for example acute anterior uveitis
- Vitreous haemorrhage

**Management by Optometrist**

**Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere**

- **Non pharmacological:** None
- **Pharmacological:** None

**Management Category**
- **A1:** emergency referral to ophthalmologist, no intervention. Telephone on-call ophthalmologist

**Possible management by Ophthalmologist**
- Admission to hospital
- Ultrasound scan
- Anterior chamber/vitreous tap, or vitrectomy (see evidence base), followed by microbiology of specimen
- Antibiotics: topical, subconjunctival, intravitreal, systemic as indicated
- Steroids: topical, intravitreal, systemic as indicated

**Evidence base**

**Sources of evidence**


**LAY SUMMARY**
The most frequently performed eye operation is cataract surgery and it is normally highly successful in restoring vision. However, in a very small proportion of cases (fewer than three per thousand) it is complicated by infection (endophthalmitis), usually caused by common bacteria.
such as those on the patient's own skin. Endophthalmitis (which means inflammation inside the eye) can also occasionally be caused by retained surgical material (e.g. cotton fibres). It is called 'acute' if it occurs within the first week after surgery and 'chronic' if it occurs up to a month after surgery. It occurs more often in patients who are diabetic or who have an infection or drug treatment that suppresses the immune system.

Endophthalmitis causes pain, redness, undue light sensitivity and blurred vision when it is acute; symptoms are less severe when it is chronic. The signs seen by the optometrist or the ophthalmologist are typical of inflammation within the eye.

If the optometrist suspects endophthalmitis, the recommendation is emergency (same day) referral to an ophthalmologist, who will usually admit the patient to hospital. A specimen is taken from within the eye so that the infecting organism can be identified and antibiotic is placed directly inside the eye. Sometimes the vitreous (the jelly inside the eye) is removed. Antibiotics may also be given as eye drops, injections beneath the skin of the eye, and by mouth or by infusion into a vein.
### Aetiology

Inward rotation of the tarsus and lid margin, causing the lashes to come into contact with the ocular surface. Most cases have a single aetiology but in some are multi-factorial

- **Involutional (age-related)**
  - Commonest cause of entropion, affects lower lid (occurs in approx. 2% of the elderly population)
  - Results from a combination of age related degenerations
    - horizontal lid laxity resulting from thinning and atrophy of the tarsus and the canthal tendons
    - weakness of the lower lid retractors
    - overriding of the preseptal over the pre-tarsal portion of the orbicularis oculi muscle, at the lid margin. This causes inward rotation of the tarsal plate on lid closure

- **Cicatricial**
  - Severe scarring and contraction of the palpebral conjunctiva pulls the lid margin inwards (ocular cicatricial pemphigoid, Stevens-Johnson syndrome, trachoma, chemical burns, post-operative complication)

- **Spastic**
  - Caused by spastic contraction of the orbicularis muscle triggered by ocular irritation (including surgery) or due to essential blepharospasm. Usually resolves spontaneously once the cause has been removed

- **Congenital**
  - Very rare entropion of the lower lid due to improper attachment of the retractor muscles to the inferior border of the tarsal plate

### Predisposing factors

- Age-related degenerative changes in the lid
- Severe cicatrising disease affecting the tarsal conjunctiva
- Ocular irritation or previous surgery

### Symptoms

- Foreign body sensation, irritation
- Red, watery eye
- Blurring of vision

### Signs

- Corneal and/or conjunctival epithelial disturbance from abrasion by the lashes (wide range of severity)
- Localised conjunctival hyperaemia
- Lid laxity (involutional entropion)
- Conjunctival scarring (cicatricial entropion)
- Absence of lower lid crease (congenital entropion)

**Distraction test**
- if lower lid can be pulled >6mm from globe, it is lax
- positive test indicates canthal tendon laxity

**Snap-back test**
- with finger, pull lower lid down towards inferior orbital margin
- release: lid should snap back
- positive test indicates poor orbicularis tone

### Differential diagnosis

Eyelid retraction (e.g. Graves’ disease):
- retracted upper or lower lid causes the lashes to be hidden by the
Entropion

resulting fold of lid skin, resembling entropion

Distichiasis:
- congenital additional row of lashes at the meibomian gland orifices

Trichiasis:
- lashes arise from normal position but are misdirected towards the cornea, secondary to inflammation

Dermatochalasis:
- degenerative condition, common in the elderly, leading to baggy appearance due to redundant lid skin and protrusion of orbital fat. Misdirection of lashes of upper lid may resemble entropion

Epiblepharon:
- congenital condition in which a fold of skin and muscle extends horizontally across the lid margin causing the lashes to be directed vertically. Orientation of tarsal plate normal. Usually asymptomatic and resolves with increasing age

Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

Non pharmacological

Taping the lid to the skin of the cheek, so as to pull it away from the globe, can give temporary relief (particularly for involutional or spastic entropion)

Epilation of lashes can be done where the trichiasis is localised (eg in cicatricial entropion)

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Therapeutic contact lens to protect cornea from lashes

(GRADE*: Level of evidence=low, Strength of recommendation=weak)

Pharmacological

Ocular lubricants for tear deficiency/instability related symptoms (drops for use during the day, unmedicated ointment for use at bedtime)

NB Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Clinical Management Guideline on Conjunctivitis Medicamentosa). They should be switched to unpreserved preparations

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Management Category

B1: Initial management (including drugs) followed by routine referral

Congenital entropion does not resolve spontaneously and the potential for severe corneal complications requires referral for prompt treatment

Possible management by Ophthalmologist

The choice of surgical procedure depends on the underlying cause(s)

Surgical intervention is indicated if any of the following are persistent:
- ocular irritation
- recurrent bacterial conjunctivitis
- reflex tear hypersecretion
- superficial keratopathy
- risk of ulceration and microbial keratitis

There is evidence that the combination of horizontal and vertical eyelid tightening is an effective treatment for involutional entropion
Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence

Boboridis KG, Bunce C. Interventions for involutional lower lid entropion. Cochrane Database Syst Rev. 2011;(12):CD002221


LAY SUMMARY

Entropion is a condition in which the edge of the eyelid (usually the lower lid) rolls inwards, so that the eyelashes touch the surface of the eye. The commonest cause is loss of elasticity and muscle tone of the eyelids which happens as part of the ageing process. It can also result if the eyelid is scarred following inflammation or injury. In many countries of the world entropion occurs as a complication of repeated infection by the trachoma agent (Chlamydia trachomatis).

The affected eye becomes irritable, red and watery, and vision may be blurred. The optometrist will be able to see the effect of eyelashes rubbing on the eye surface and may be able to determine the cause. Taping the edge of the eyelid to the skin of the cheek may give temporary relief, as may the removal of lashes or the fitting of a bandage contact lens to protect the eye surface from contact with the eyelashes. Patients may be helped by artificial tears and unmedicated ointments. These measures will not cure the condition, so patients are often referred routinely to the ophthalmologist for consideration of surgery, usually under local anaesthetic, which may solve the problem.
### Episcleritis

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Idiopathic inflammation of the vascular connective tissue layer that lies between the sclera and conjunctiva</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predisposing factors</td>
<td>Up to one third of cases (especially nodular variety) associated with systemic disorders, e.g.</td>
</tr>
<tr>
<td></td>
<td>• collagen vascular diseases</td>
</tr>
<tr>
<td></td>
<td>• rheumatoid arthritis</td>
</tr>
<tr>
<td></td>
<td>• systemic lupus erythematosus</td>
</tr>
<tr>
<td></td>
<td>• inflammatory bowel disease</td>
</tr>
<tr>
<td></td>
<td>Also other conditions including gout, ocular rosacea</td>
</tr>
<tr>
<td></td>
<td>(NB importance of careful history taking)</td>
</tr>
<tr>
<td></td>
<td>Previous history of episcleritis</td>
</tr>
<tr>
<td></td>
<td>Herpes Zoster Ophthalmicus</td>
</tr>
<tr>
<td></td>
<td>Commonest in 4th or 5th decades</td>
</tr>
</tbody>
</table>

| Symptoms          | Acute onset                                                                                            |
|                   | Typically unilateral red eye, but bilateral in a quarter to a half of cases                            |
|                   | Mild ache or burning sensation                                                                          |
|                   | Sometimes tender on palpation                                                                           |
|                   | Occasionally watery                                                                                    |
|                   | Condition commonly recurrent                                                                          |

| Signs              | Hyperaemia from dilated episcleral vessels (NB these follow a regular radiating pattern and are immovable, unlike the finer overlying conjunctival vessels which move freely with the conjunctiva). |
|                   | Hyperaemia blanches with vasoconstrictors (e.g. gutt. phenylephrine 10%)                             |
|                   | Simple (80%)                                                                                           |
|                   |   • sectoral or diffuse redness                                                                         |
|                   | Nodular (20%)                                                                                          |
|                   |   • nodule (mild elevation of the conjunctiva) with injection                                          |
|                   | Typically no anterior chamber reaction                                                                |
|                   | Usually no corneal or palpebral conjunctival involvement                                               |
|                   | No effect on visual acuity                                                                            |

| Differential diagnosis | Scleritis (see Clinical Management Guideline on Scleritis)                                               |
|                       | Conjunctivitis (viral, bacterial, allergic)                                                              |
|                       | Phlyctenular keratoconjunctivitis                                                                     |
|                       | Pingueculitis                                                                                            |
|                       | Anterior uveitis                                                                                       |

### Management by Optometrist

**Practice guidelines**

**Non pharmacological**

- Usually self-limiting in 7-10 days
- Reassurance: condition does not generally progress to more serious ocular disorder
- Cold compresses
- Advise patient to return/seek further help if symptoms persist
  
  (GRADE*: Level of evidence=low, Strength of recommendation=strong)

**Pharmacological**

- Mild cases: no specific treatment
- If discomfort: artificial tears as necessary for 1-2 weeks
  
  (GRADE*: Level of evidence=low, Strength of recommendation=strong)
- Inconsistent evidence for benefit of topical NSAIDs (off-licence use)
  
  (GRADE*: Level of evidence=moderate, Strength of recommendation=weak)
### Episcleritis

| Management Category | B3: management to resolution
|                     | B2 (modified): episcleritis with symptoms suggestive of systemic disease, or at second recurrence (third episode), refer for investigation

| Possible management by Ophthalmologist | Investigation for underlying systemic disease

| Evidence base |
|---------------|----------------------------------|
| *GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm) |

**Sources of evidence**


**LAY SUMMARY**

Episcleritis is an inflammation of the episclera, the tissue that lies just under the outer skin of the white of the eye. Commonest between the ages of 40 to 60, it usually affects just one eye but both eyes are affected in a quarter to a half of cases. In about one in three of cases there is a background of inflammation elsewhere in the body, for example rheumatoid arthritis or inflammation of the bowel.

The condition begins without warning and patients experience redness, aching and tenderness of one or both eyes. The vision is not affected. Episcleritis may disappear in a week to ten days and return again later. The optometrist will reach a diagnosis based on the exact type of inflammation, which distinguishes this condition from conjunctivitis (inflammation of the outer skin of the eye) and scleritis (inflammation of the white part of the eyeball).
Episcleritis

Cold compresses may relieve the symptoms, as may artificial tears in eye drop form. If the inflammation is more severe, steroid eye drops may be prescribed, and sometimes anti-inflammatory tablets are needed also.

After the second recurrence, the optometrist will normally refer the patient for investigation by the ophthalmologist.
### Aetiology

Paralysis of facial nerve (VII cranial nerve)

This Clinical Management Guideline addresses Bell’s Palsy (idiopathic lower motor neurone facial nerve dysfunction), which constitutes 72% of all facial palsy:

- annual incidence 20-30 per 100,000, especially between 15 & 45 yrs with a possible second peak in the over-70s
- sudden onset, unilateral
- M = F (except in pregnancy, see below)
- cause unknown, but sometimes associated with latent virus infection (HSV type 1, Herpes zoster)
- fair prognosis without treatment
  - however, up to 30% of treated patients do not recover completely
- better prognosis with systemic steroid therapy (with or without anti-viral)
- most improvement occurs within three weeks

Other causes of paralysis of the facial nerve, which are not addressed in this Clinical Management Guideline, include:

- infection, e.g. otitis media
- trauma, e.g. temporal bone fracture
- tumour compressing the facial nerve, e.g. acoustic neuroma
- sarcoidosis
- Guillain-Barré syndrome
- cerebrovascular accident (stroke)

### Predisposing factors

Bell’s Palsy is more common in:

- pregnancy (annual incidence increases to 45 per 100,000)
- diabetes
- HIV

### Symptoms

Distressing cosmetic change due to loss of muscle tone on one side of face

Watering of eye (epiphora)

Ocular exposure causes:

- redness, discomfort, pain, photophobia, reduced vision

### Signs

Unilateral facial weakness including orbicularis oculi

- incomplete blink leads to corneal drying
- incomplete lid closure at night (lagophthalmos) causes prolonged corneal exposure
- loss of lacrimal pump mechanism produces pooling and epiphora

Conjunctival hyperaemia, oedema, staining

Corneal desiccation ranges from mild superficial punctate erosions to frank ulceration (usually inferior)

### Differential diagnosis

Other causes of facial nerve palsy:

- as part of a stroke (cerebro-vascular accident with hemiplegia), in which forehead muscle function is preserved
- infection (e.g. otitis media, Lyme disease)
- trauma (e.g. cranial fracture, facial laceration)
- tumour (e.g. acoustic neuroma: damage to nerve by tumour or secondary to surgical trauma; also perineural invasion by a squamous cell carcinoma)

Ectropion or Entropion
Facial Palsy (Bell’s Palsy)

Other causes of lagophthalmos
- orbital (thyroid eye disease – assess by exophthalmometry)
- mechanical (cicatricial – look for lid scarring)
- physiological (patient’s partner to check for full lid closure at night)

Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

| Non pharmacological | Tape lids closed at night (and during day if corneal desiccation is marked)
GRADE*: Level of evidence=low, Strength of recommendation=strong |
| Sunglasses for photophobia and general protection
GRADE*: Level of evidence=low, Strength of recommendation=strong |
| Therapeutic contact lens (if unresponsive to frequent use of ocular lubricants)
- silicone hydrogel should be considered as first choice (however, scleral lens gives maximum protection)
NB therapeutic contact lens fitting is contraindicated in cases of neurotrophic keratitis with loss of corneal sensation (cranial nerve V). Such patients are at high risk of infection, which may be further increased by contact lens wear
GRADE*: Level of evidence=low, Strength of recommendation=weak |
| Pharmacological | Tear supplements / lubricants by day, unmedicated ointment at night
GRADE*: Level of evidence=low, Strength of recommendation=strong |

Management Category

New cases, and where there is loss of corneal sensation:
A2: first aid measures and emergency (same day) referral to GP or ophthalmologist
Improved prognosis in moderate/severe cases of Bell’s palsy if treated with systemic corticosteroid (with or without anti-viral) within 72 hours of onset
NB: corneal ulceration due to exposure is potentially sight threatening and may justify emergency referral

Recovering and established cases:
B2: alleviation/palliation; no referral
If cannot be managed easily, then:
B1: prescription of drugs; routine referral

Possible management by Ophthalmologist

Urgent medical treatment for new cases:
- systemic steroid ± anti-viral
Temporary tarsorrhaphy
Upper lid lowering with botulinum toxin injection of levator muscle
Surgery for permanently unrecovered cases:
- tarsorrhaphy (permanent)
- upper lid lowering (surgery, gold weight)
- surgical sling to raise lower lid

Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)
Facial palsy (Bell’s Palsy)

Sources of evidence


LAY SUMMARY

Facial palsy results if the nerve supplying the muscles of the face, including the circular muscle around the eye, stops functioning. There are many causes, but Bell’s Palsy is the commonest, accounting for nearly three quarters of all cases. Usually this affects only one side of the face and is temporary, lasting around three weeks, though recovery may not be complete. The cause is unknown. People between the ages of 15 and 45 are most likely to be affected, but the condition is commoner in those who are pregnant, have diabetes or are living with HIV infection.

Patients notice that the affected side of the face droops and does not move. The eye may not close properly and as a result it can become red, uncomfortable and watery. The optometrist will examine the eye for signs of drying and for loss of feeling, which sometimes occurs. New cases will be referred as emergencies to the GP or the ophthalmologist, as recovery is improved if steroid tablets are given within 72 hours of the onset of symptoms. Longer-standing cases are managed by the optometrist and if necessary referred routinely to the ophthalmologist.

The optometrist will usually prescribe artificial tears to be used frequently during the day and ointment at night. Taping the eyelids closed at night may help. Sunglasses will often relieve light sensitivity and physically protect the eye. Sometimes a contact lens will be fitted to protect the cornea (the clear window of the eye).
### Aetiology
Patient may give history of foreign body entering eye
- particle falling into eye (rust while working under car, DIY debris)
- wind blown from unknown source

### Predisposing factors
Lack of suitable eye protection

### Symptoms
Foreign body sensation / acute pain
Lacrimation
Red eye

### Signs
Possible fluorescein staining of cornea
- foreign body tracks, often vertical
Embedded material on tarsal conjunctival surface
Hyperaemia

### Differential diagnosis
- Dendritic ulcer
- Corneal abrasion (e.g. from contact lens)
- Superficial punctate keratopathy of another cause
*NB: check cornea for adherent/embedded material*

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

#### Non pharmacological
Evert upper eyelid (may require double eversion)
Remove foreign body, after instillation of topical anaesthetic, with:
- saline irrigation
- saline-wetted cotton bud (can also be used to sweep the fornix)
- sterile hypodermic needle if cannot be dislodged by cotton bud
*(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

Advise patient to return/seek further help if symptoms persist
Advise on future suitable eye protection
*(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

#### Pharmacological
Local anaesthetic (e.g. g. oxybuprocaine 0.4%) to aid examination and removal of foreign body
*(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

After removal, prescribe tear supplements / lubricants for symptomatic relief
*(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

Consider prophylactic antibiotic (e.g. course of chloramphenicol drops/ointment for not less than 5 days) if there is substantial epithelial loss or foreign matter contamination of the conjunctival sac (see Clinical Management Guideline on Corneal Abrasion)
*(GRADE*: Level of evidence=low, Strength of recommendation=weak)*

### Management Category
**B3:** Management (including drugs) to resolution. Normally no referral

### Possible management by Ophthalmologist
(Normally no referral)

### Evidence base

Sources of evidence
None applicable
LAY SUMMARY

Sometimes a speck of dust or other debris, landing on the eye, becomes trapped under the upper eyelid. While there it may cause discomfort which increases on blinking when the foreign body is rubbed over the eye surface. Usually the patient will know that something has blown into the eye.

The optometrist will turn the upper eyelid over and, if a foreign body is found, remove it. Sometimes a drop of local anaesthetic is needed to reduce the discomfort of this procedure. Generally, no further treatment is needed, but if there is a large abrasion of the surface of the cornea (the clear window at the front of the eye) or any evidence of infection, a course of local antibiotic drops or ointment may be prescribed. Such cases are not usually referred to the ophthalmologist.
Primary Angle Closure / Primary Angle Closure Glaucoma (PAC / PACG)

**Aetiology**

Primary angle closure (PAC) is defined as appositional or synechial closure of the anterior chamber angle which can lead to aqueous outflow obstruction and raised IOP, in the absence of glaucomatous optic neuropathy. PAC is generally bilateral.

Optic nerve damage resulting from PAC is described as primary angle closure glaucoma (PACG). The pooled prevalence of PACG among Caucasians of European ancestry aged 40 and over is 0.4%

PACG is caused by a variety of mechanisms although pupil block, in which aqueous is impeded on its passage between the lens and posterior surface of the iris, is considered to be a key element in its pathogenesis.

Patients with angle closure disease may be categorized as follows:

<table>
<thead>
<tr>
<th>Feature</th>
<th>PAC Suspect</th>
<th>PAC</th>
<th>PACG</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥180 degrees ITC</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Elevated IOP and/or PAS</td>
<td>Absent</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Optic nerve damage</td>
<td>Absent</td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>

(ITC = irido-trabecular contact, PAS = peripheral anterior synechiae)

Acute angle closure crisis (AAC): typically PAC and PACG develop chronically without symptoms, however an acute rise in IOP (unilateral in 90% of cases) can present as a clinical emergency.

**Predisposing factors**

**Anatomical**

Associated with:
- sex (F:M ratio 3:1)
- ethnicity (e.g. Chinese, Vietnamese, Inuit). PACG is recognized as a leading cause of blindness in East Asia
- family history
- short axial length (hypermetropia)
- shallow AC (F>M)
- increasing age (AC becomes shallower as lens thickness increases)
- small corneal diameter

**Iatrogenic** (secondary angle closure)

- Drug induced (topical and systemic, see Evidence Base)
  Adrenergic agents e.g. phenylephrine
  Drugs with anticholinergic effects e.g. tricyclic antidepressants
  Drugs that may cause ciliary body oedema, e.g. topiramate, sulphonamides
- Surgery induced
  Angle closure may follow a number of surgical procedures, for example vitreo-retinal surgery with intraocular gas, especially in aphakic eyes

**Symptoms**

Patients with PAC can be asymptomatic or mildly symptomatic (ocular discomfort, headache). AAC is associated with sudden onset of symptoms and signs:
- rapid progressive impairment of vision of one or both eyes
Primary Angle Closure / Primary Angle Closure Glaucoma (PAC / PACG)

- ocular and periocular pain which can be severe
- nausea and vomiting
- ocular redness

50% of patients with an acute angle closure attack give a history of previous intermittent attacks, e.g. episodes of blurring of vision lasting 1-2 hours, associated with haloes around lights, eye ache or frontal headache

**Signs**

In a PAC suspect the eye may appear normal (with the exception of a narrow angle, as judged by the van Herick technique or by gonioscopy) In cases with a narrow van Herick angle (≤ 25% [Grade 1 or 2]) with a normal anterior chamber depth, plateau iris should be suspected

In AAC the following signs may be present:

- limbal and conjunctival vessels dilated, producing ciliary flush
- pupil fixed, semi-dilated, vertically elliptical, iris whirling
- corneal oedema
- shallow AC with peripheral irido-corneal contact (if angle can be visualised)
- high intraocular pressure (40-80mmHg)
- AC flare and cells
- optic disc oedematous and hyperaemic
- grey/white anterior sub-capsular lenticular opacities (Glaukomflecken): diagnostic of previous attacks

**Differential diagnosis**

- Neovascular glaucoma
- Phakolytic glaucoma
- Phakomorphic glaucoma
- Acute anterior uveitis
- Uveitis with raised IOP
- Malignant glaucoma (cilio-lenticular block or aqueous misdirection glaucoma)

**Management by Optometrist**

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

**Non-pharmacological**

**Potentially occludable angle as judged by van Herick test**

NICE does not provide guidance on referral for angle closure; however SIGN recommends that patients with peripheral anterior chamber width of ≤25% of the corneal thickness (van Herick Grade 1 or 2) should be referred to secondary eyecare services

*(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

**PAC Suspect**

Can only be diagnosed by gonioscopy. The decision to refer for further treatment should be based on the risk of developing PAC/PACG or AAC. If not referring for further investigation, patients with PACS require close monitoring and serial gonioscopy. Patients should be aware that they are at risk of occlusion and that certain medications could induce angle closure

*(GRADE*: Level of evidence=low, Strength of recommendation=strong)*
Primary Angle Closure / Primary Angle Closure Glaucoma (PAC / PACG)

<table>
<thead>
<tr>
<th>PAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>The current clinical consensus is that patients with PAC/PACG should be treated surgically (peripheral iridotomy or cataract extraction) to relieve pupillary block together with pharmacological therapy to reduce elevated IOP (GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

**Pharmacological**

<table>
<thead>
<tr>
<th>AAC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior to referral, commence first aid treatment with a drop of pilocarpine 2% eye drops in blue eyes and 4% eye drops in brown eyes (although this is likely to be ineffective when IOP is over 40mmHg and paradoxically pilocarpine can exacerbate angle closure by inducing anterior lens movement) (GRADE*: Level of evidence=low, Strength of recommendation=weak)</td>
</tr>
<tr>
<td>Where the IOP is 40mmHg or higher and the patient is not vomiting, give a single dose of oral acetazolamide (Diamox) 500mg (not slow release formulation). (NB: Diamox may be hazardous in an elderly frail patient.) Then refer as an emergency to ophthalmologist. (In view of potential unwanted effects of this treatment, patient should be accompanied by a carer or relative) (GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

**Management Category**

| A2: first aid measures and emergency (same day) referral to ophthalmologist |
| PAC/PACG |
| A3: urgent (within one week) referral to ophthalmologist; no intervention |
| PAC Suspect |
| A3 (modified): routine referral to ophthalmologist; no intervention |

**Possible management by Ophthalmologist**

<table>
<thead>
<tr>
<th>AAC: treatment directed to breaking the pupil block and reducing IOP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical</td>
</tr>
<tr>
<td>• miotics (e.g. gutt. pilocarpine 2-4%)</td>
</tr>
<tr>
<td>• systemic agents (e.g. acetazolamide, glycerol)</td>
</tr>
<tr>
<td>• topical antihypertensives (e.g. gutt. timolol, gutt. dorzolamide, gutt. brimonidine)</td>
</tr>
<tr>
<td>Urgent interventions</td>
</tr>
<tr>
<td>• anterior chamber paracentesis (occasionally used in advance of peripheral iridotomy)</td>
</tr>
<tr>
<td>• argon laser peripheral iridoplasty (occasionally used in advance of YAG laser peripheral iridotomy [LPI])</td>
</tr>
<tr>
<td>• LPI</td>
</tr>
<tr>
<td>Less urgent interventions</td>
</tr>
<tr>
<td>• cataract surgery</td>
</tr>
<tr>
<td>• clear lens extraction</td>
</tr>
<tr>
<td>• selective laser trabeculoplasty, post LPI</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PAC / PACG: first line treatment options include:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• topical medical therapy</td>
</tr>
<tr>
<td>• LPI (for patients with PACG)</td>
</tr>
</tbody>
</table>
Primary Angle Closure Glaucoma (PACG) is rarer in this country than Primary Open Angle Glaucoma, and in its acute form differs in that the drainage route for the fluid inside the eye is closed off, rather than gradually blocked. It affects women more often than men, is commoner in...
Primary Angle Closure / Primary Angle Closure Glaucoma (PAC / PACG)

long-sighted people and people of East Asian ancestry, and becomes more likely to occur as people age. Certain drugs and eye operations can also cause the drainage angle to close. A sudden complete closure of the drainage route (known as acute angle closure crisis, AAC), which usually affects just one eye, causes rapidly progressing impairment of vision, redness of the eye, and pain in and around the eye which may be so severe as to cause nausea and vomiting. The eye pressure may be very high, because the fluid continues to be formed within the eye but cannot drain away. Various other changes will be seen in the eye by the examining optometrist. An acute attack of angle closure is an emergency which needs same-day referral to the ophthalmologist. There are drugs that the optometrist can use as first aid. The ophthalmologist will also prescribe drugs and may advise laser treatment to create a tiny hole in the iris (the coloured part of the eye) through which the fluid can drain. If at a routine eye examination there are signs that there have been earlier, milder attacks of angle closure, or if it appears that a patient could develop PACG, the referral can be urgent, or may be made with less urgency.
### Aetiology

Primary Open Angle Glaucoma is a progressive optic neuropathy associated with a loss of retinal ganglion cells and their axons. The anterior chamber angle is open and there is no secondary cause for the optic nerve damage, e.g. steroid glaucoma, pseudoexfoliative or pigmentary glaucoma.

Affects 1-2% of the white population of the UK aged over 40 years, increasing to 4-5% in the over-80s. Second most common cause of irreversible blindness in the UK (approx. 10% of blind registrations).

### Predisposing factors

- Ocular hypertension
- Increasing age
- Positive family history in a first degree relative
- People of West African descent including West Indians and African Americans; onset at younger age
- Thinner central cornea
- Myopia > 3D (odds ratio = 2.46)
- Diabetes (odds ratio = 1.35)
- Systemic hypertension

### Symptoms

Usually asymptomatic until there is significant loss of visual field.

### Signs

**Optic disc:**
- cupping:
  - generalised thinning, focal narrowing or notching of neuroretinal rim
  - cupping should be considered in relation to disc size
  - CD ratio >0.6 or R:L asymmetry ≥ 0.2 suspicious of glaucoma
- disc margin haemorrhage is an important prognostic sign but not necessarily diagnostic of glaucoma (more common in Normal Tension Glaucoma)
- defects of the nerve fibre layer visible in younger patients
- if available, establish baseline using stereo-photography or with computer-based image analysis e.g. confocal scanning laser ophthalmoscope (CSLO) or ocular coherence tomography (OCT)

**Visual fields:**
- reproducible visual field test defect consistent with optic disc appearance
- relative or absolute arcuate scotoma, nasal step, paracentral loss

**Applanation tonometry:**
- IOP >21mmHg
  - greater central corneal thickness produces artefactually high IOP measurements
  - lesser central corneal thickness produces artefactually low IOP measurements

**NB:** In a significant proportion of patients with glaucoma, IOP is in normal range (Normal Tension Glaucoma)
- greater than normal diurnal variation in IOP (>4 mm Hg)
  - measure IOP at different times of day; usually highest in morning

**Gonioscopy:**
Glaucoma (primary open angle) (POAG)

- open angles with normal appearance
  - Shaffer Grading 3 or 4

Assessment of anterior chamber angle depth at slit lamp:
- van Herick Grading (limbal anterior chamber depth >25% of corneal thickness [Grade 3 or 4])

<table>
<thead>
<tr>
<th>Differential diagnosis</th>
<th>Ocular hypertension (OHT)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>defined as intraocular pressure above the normal range, without anterior segment abnormality, optic nerve cupping or visual field loss</td>
</tr>
<tr>
<td></td>
<td>around 10% of ocular hypertensives will develop glaucoma in 5 years</td>
</tr>
<tr>
<td></td>
<td>(See Clinical Management Guideline on Ocular Hypertension)</td>
</tr>
<tr>
<td></td>
<td>Tilted optic discs, physiological cupping, disc drusen, anterior ischaemic optic neuropathy</td>
</tr>
<tr>
<td></td>
<td>Secondary glaucoma masquerading as POAG (e.g. steroid responder, pigment dispersion, pseudo-exfoliation)</td>
</tr>
<tr>
<td></td>
<td>Primary angle closure (PAC) / Primary angle closure glaucoma (PACG)</td>
</tr>
</tbody>
</table>

Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

Non pharmacological

Guidance on glaucoma case finding is included in National Institute Health and Care Excellence (NICE) guideline (NG81, November 2017): Glaucoma: diagnosis and management; Scottish Intercollegiate Guidelines Network guideline (SIGN 144, March 2015): ‘Glaucoma referral and safe discharge’ (see Evidence Base); and guidance produced by the College of Optometrists: ‘Examining patients at risk from glaucoma’. NICE guideline NG81 also provides detailed guidance on the diagnosis and management of POAG (including monitoring intervals).

Diagnosis: refer people with suspected optic nerve damage or repeated visual field defect (or both) to a consultant ophthalmologist for consideration of a definitive diagnosis and formulation of a management plan (NICE recommendation).

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

People with POAG should have monitoring and treatment from a trained healthcare professional who has all of the following: a specialist qualification in glaucoma; relevant experience; ability to detect a change in clinical status. Holding an independent or non-medical prescribing qualification alone (without a specialist qualification relevant to the case complexity of glaucoma being managed) is insufficient for managing glaucoma and related conditions (NICE recommendation).

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Offer people the opportunity to discuss their diagnosis, referral, prognosis, treatment and discharge, and provide them with relevant information in an accessible format at initial and subsequent visits. (NICE recommendation)

(GRADE*: Level of evidence=low, Strength of recommendation=strong)
Pharmacological

Diagnosis: do not prescribe anti-glaucoma drugs prior to assessment by a consultant ophthalmologist (NICE recommendation) (GRADE*: Level of evidence=low, Strength of recommendation=strong)

Follow-up management: people with a diagnosis of POAG can be monitored (and treated) by optometrists with a specialist qualification in glaucoma (NICE recommendation) (GRADE*: Level of evidence=low, Strength of recommendation=strong)

Management Category

A3 (modified): In England, unless clinical circumstances indicate that urgent or emergency referral is indicated, patients should have referral filtering (e.g. repeat measures, referral refinement) before they are referred to the HES. In Scotland, referral criteria should follow SIGN guidelines

Possible management by Ophthalmologist

Confirmation of diagnosis of POAG
Determination of the individual clinical management plan
Reduce IOP pharmacologically taking into account:
• estimated target IOP for the individual patient
• likely degree of compliance (the simpler, the better)
• side effects, contra-indications and drug interactions

Most POAG is treated with eye drops
• prostaglandin analogues are first choice
• beta-blockers are second choice (relatively high incidence of unwanted effects)
• other choices are carbonic anhydrase inhibitors and alpha agonists
• Selective Laser Trabeculoplasty (SLT) is increasingly used as a first line treatment
• systemic treatment for POAG is rarely needed. Long-term therapy with oral carbonic anhydrase inhibitors may be necessary in a few refractory cases but drug intolerance is common

Surgery (incisional or non-incisional) may be required

Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)

Sources of evidence


### Table of References

<table>
<thead>
<tr>
<th>Reference</th>
</tr>
</thead>
</table>

### LAY SUMMARY

Primary Open Angle Glaucoma (POAG) affects approximately 1-2% of the white population of the UK over 40 years of age, increasing to 4-5% of those over 80 years. It is more likely to affect people with a family history of the same condition, and people of West African ancestry, including West Indians and African Americans. The condition is not painful and patients may be unaware that they have it until they have started to lose vision.

The optometrist examining a patient for POAG, will look for the characteristic appearance of ‘cupping’ of the optic disc (the head of the optic nerve at the back of the eye). The optic nerve transfers visual information from the eye to the brain, and can be examined with various special instruments. On testing the patient’s fields of vision, defects may be found that are typical of glaucoma. The pressure of the fluid inside the eye (known as the intraocular pressure, or IOP) must be measured as it is usually raised in glaucoma. In a small proportion of patients, however, the pressure is within the normal range.

The eyeball is a sphere kept inflated by fresh clear fluid formed within the eye. This fluid drains away through a fine ring-shaped sieve of tissue, known as the drainage angle (or simply angle), situated within the eye at the edge of the cornea (the clear window of the eye) and from there into the bloodstream. This sieve is not directly visible but it can be seen through a special contact lens containing a mirror. In POAG the fluid is unable to escape normally because the meshwork has become blocked. The blockage causes the pressure inside the eye to rise, and in time this can damage the optic nerve, causing loss of visual field and even blindness if the condition is not treated. Treatment is usually with eye drops, which slow the production of fluid or increase its drainage, but if these are inadequate an operation may be needed.

The optometrist who discovers POAG will usually refer the patient routinely to the ophthalmologist. Sometimes POAG develops out of another condition called Ocular Hypertension, for which a separate Clinical Management Guideline has been written.
**Aetiology**

Elevation of IOP has been associated with both ocular and systemic administration of steroids (e.g. topical application to the eye or eyelids, sub-Tenon's injection, intra-vitreal injection [or implant], steroid by mouth).

If the elevated IOP is of sufficient magnitude and duration, damage to the optic nerve can occur with resulting visual field loss (steroid glaucoma).

The mechanism of elevated intraocular pressure is increased aqueous outflow resistance owing to an accumulation of extracellular matrix material in the trabecular meshwork.

With regard to this 'steroid response' the normal population can be divided into three groups:

- High responders (5%): marked elevation of IOP by >16mmHg
- Moderate responders (30%): moderate elevation by 6-15mmHg
- Non-responders (65%): an elevation of up to 5mmHg is considered irrelevant in this instance.

The higher the steroid potency, the greater the ocular hypertensive response. The ophthalmic steroids dexamethasone and prednisolone acetate are more likely to result in clinically significant increases in IOP when compared to fluorometholone and loteprednol.

**Predisposing factors**

Primary open angle glaucoma (POAG)
- First degree relative with POAG
- Childhood
- High myopia
- Diabetes

**Symptoms**

None in the early stages; visual loss later.

**Signs**

Raised intraocular pressure following use of topical steroid.

If optic neuropathy is present, the condition clinically resembles POAG (although higher IOP compared to POAG, resulting in more rapid visual field and optic disc changes).

Can occur at any time (within weeks with potent drugs and after several months with weaker agents); onset rare with less than 3 weeks’ exposure.

**Differential diagnosis**

- POAG
- Ocular hypertension
- Secondary glaucoma (pigment dispersion, pseudo-exfoliation, neovascular, inflammatory [e.g. Posner-Schlossman syndrome or following anterior uveitis])

**Management by Optometrist**

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.

**Non pharmacological**

A baseline measurement of IOP should always be taken prior to commencement of steroid therapy. Patients newly begun on ocular steroid therapy should have their IOPs measured again after 2 weeks, then every 4 weeks for 2-3 months, then 6-monthly if therapy is to continue.

It has been recommended in patients receiving intravitreal steroids (injections or implants) to measure IOP at 30 minutes, at 1 week, 2 weeks and monthly for up to 6 months.

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**Glaucoma (steroid)**

Version 11, Page 1 of 3

Date of search 20.12.17; Date of revision 22.02.18; Date of publication ab.cd.ef; Date for review 19.12.19

© College of Optometrists
If a steroid response is detected, discuss with the prescribing clinician the possibility of discontinuing steroid therapy (the chronic steroid response usually resolves in 1-4 weeks, whilst the acute response may resolve within a few days of cessation of steroid therapy)

| Pharmacological | Patient may need topical anti-glaucoma medication (discuss with prescribing clinician)  
GRADE*: Level of evidence=low, Strength of recommendation=strong |
| Management Category | **A3**: possible first aid measures followed by urgent referral to an ophthalmologist (within one week)  
If the optometrist is the prescribing clinician, discontinue steroid or switch to 'non-penetrating' steroid and monitor IOP until it reaches an acceptable level. Patient may need short-term ocular hypotensive medication. In this situation, referral may not be necessary  
**B3**: management to resolution |
| Possible management by Ophthalmologist | Alternative therapy with topical steroids that are less likely to cause raised IOP and/or with NSAIDs  
Anti-glaucoma management (medical ± surgical including laser) |
| Evidence base | GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)  
Sources of evidence  
Pleyer U, Ursell PG, Rama P. Intraocular pressure effects of common...
LAY SUMMARY

The pressure inside the eye can rise if a patient is treated with steroids, either as eye drops, injection into the eye tissues, tablets or by injection. 35% of the population is at risk of developing this problem. If the raised pressure is not identified, glaucoma can result. This is a disease in which raised eye pressure can damage the nerve fibres of the retina (the light-sensitive layer at the back of the eye) and cause selective but irreparable loss of the eye’s field of vision.

Stopping the steroid that causes the condition, if this is medically possible, usually allows the eye pressure to return to normal. If this does not happen, the condition may have to be treated as if it were an ordinary case of glaucoma (of the type known as ‘open angle’), using eye drops or possibly laser treatment or surgery.
## Aetiology

Herpes simplex virus (HSV) infection is extremely common, though usually latent

- up to 90% of UK population is seropositive for HSV

HSV-1 generally infects ‘above the waist’ (lips, face, eyes)

- primary infection usually in childhood, then virus lies dormant in trigeminal ganglion
- when virus reactivates it travels along branches of the trigeminal nerve to cause local infection (e.g. cold sore or herpes keratitis)

HSV-2 generally infects ‘below the waist’ and is usually sexually acquired

- but may also be a cause of herpetic keratitis

Ocular HSV infection (of which the incidence of new cases is 5-15 per 100,000 per annum) can manifest as blepharoconjunctivitis, keratitis, iridocyclitis or acute retinal necrosis. The most common form is epithelial keratitis, accounting for 50% to 80% of cases

Ocular HSV infection can be categorised into primary and recurrent disease

Herpes simplex keratitis (HSK) is the leading cause of corneal blindness in developed countries. In UK, responsible for 1 in 10 corneal transplants

## Predisposing factors

- Poor general health, immunodeficiency, fatigue
- Systemic or topical steroids, or other immunosuppressive drugs
- Possible aggravating factors
  - sunlight (UV), fever, extreme heat or cold, infection (systemic or ocular), trauma (ocular)
- History of previous attacks of ocular herpes simplex infection (key diagnostic feature)
- Severe atopic disease

## Symptoms

- Usually affects one eye; may be bilateral, especially in severely atopic patients
- Severity of symptoms very variable
- Pain, burning, irritation, photophobia, reduced visual acuity, redness

## Signs

HSK has a highly variable and unpredictable course
Can be considered as a spectrum of four distinct disease entities (with differing management strategies):

### Epithelial

- Initially punctate lesions, coalescing into dendriform pattern
  - dendritic ulcer, single or multiple
  - opaque cells arranged in a stellate pattern progressing to a linear branching ulcer
  - associated with reduced corneal sensitivity
  - continued enlargement may result in an ‘amoebic’ or ‘geographic’ ulcer (especially following inappropriate use of topical steroids)

### Stromal

- Necrotic stroma, stromal infiltrates, vascularisation, scarring, uveitis and keratic precipitates, possibly raised intraocular pressure

### Disciform keratitis

- Central or eccentric zone of epithelial oedema overlying an area of
<table>
<thead>
<tr>
<th><strong>stromal thickening</strong></th>
<th>Folds in Descemet’s membrane, uveitis and keratic precipitates</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Metaherpetic ulcer (trophic keratitis)</strong></td>
<td>Due to a combination of denervation, drug toxicity, persistent defects in epithelial basement membrane</td>
</tr>
</tbody>
</table>

## Differential diagnosis
- **Herpes zoster keratitis**
- Bacterial, fungal or amoebic keratitis (NB *Dendritic keratitis in a contact lens wearer should raise the index of suspicion of an Acanthamoeba infection*).
- Healing corneal epithelial defect (e.g. abrasion): may have stellate or dendritic profile.

## Management by Optometrist
**Non pharmacological**
- Exclude viral retinitis following pupil dilatation (especially in immunocompromised patients) as this would warrant emergency (same day) referral
  - peripheral infiltrates
  - vasculitis
  - intra-retinal haemorrhages
  - vitreous inflammation

**Pharmacological**
- **Acute Herpes Simplex**: in non-contact lens wearing adults and where HSK is confined to the epithelium, commence antiviral therapy with
  - ganciclovir 0.15% ophthalmic gel (NB BAK preserved preparation only)

  (The previously available first choice therapy for HSK, aciclovir 3% eye ointment, was discontinued by GlaxoSmithKline UK Ltd in late 2018)

  NB: HSK is a potentially blinding disease and optometrists should consistently apply a low threshold for referral for this condition

  **Recurrent Herpes Simplex**: where there is:
  - a clear history of previous attacks
  - no doubt about the diagnosis and
  - only epithelial involvement

  - commence antiviral therapy (as above)

  **GRADE**: Level of evidence=moderate, Strength of recommendation=strong)

## Management Category
- **B2 (modified)**: (acute or recurrent epithelial HSK with no stromal involvement): alleviation or palliation; but refer urgently (within one week) to ophthalmologist if epithelium has not healed after seven days
- **A1** (if stroma involved, or in children or contact lens wearers, or in bilateral cases): emergency (same day) referral to ophthalmologist

## Possible management by Ophthalmologist
- Isolation and characterisation of virus from corneal swab or biopsy
- Antivirals (topical and/or systemic)
- Topical steroid
- Surgical débridement
- Penetrating keratoplasty in some quiescent cases with scarring

## Evidence base
*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*
Sources of evidence


Wilhelmus KR. Antiviral treatment and other therapeutic interventions for herpes simplex virus epithelial keratitis. Cochrane Database Syst Rev. 2015;1:CD002898

LAY SUMMARY

Up to 90% of people in the UK have an inactive nerve infection caused by herpes simplex virus type 1. This infection is usually acquired in childhood by contact with an adult. The viruses penetrate the skin and travel along the nerves of sensation to the central part of the nervous system (brain or spinal cord) where they become 'latent' and produce no symptoms. The infection cannot be eliminated and there is no protective vaccine. Another variety, herpes simplex type 2, is usually sexually transmitted, but can also affect the eye.

The infection remains inactive until some factor such as poor health, disturbance of the body’s immune system or certain drugs allow the virus to become active and then travel back down the nerves to the surface of the body. If this happens in the trigeminal nerve (the nerve that gives sensation to the head and neck), an infection of the skin such as a cold sore on the lip, or an infection of the eye surface, can occur. If the cornea (the clear window at the front of the eye) is involved, the condition is known as Herpes Simplex Keratitis. Usually only one eye is affected.

Several different forms of corneal infection are possible, ranging from slight to severe. One of them, affecting the surface skin of the cornea, produces ulcers with a characteristic branching outline and this is known as a dendritic ulcer. The infection can recur and if this happens scarring may result, which can cause blurring of vision.

A new case, with involvement of the surface skin of the cornea only, will usually be treated by the optometrist with anti-viral eye ointment. In children, contact lens wearers and where the condition affects both eyes, same-day referral to the ophthalmologist is recommended. This is a condition which tends to recur from time to time. Recurrences involving only the surface skin of the cornea can often be managed by the optometrist, who will usually prescribe anti-viral eye ointment, but if this is not effective after a week, urgent referral to the ophthalmologist is recommended. If the vision has become badly affected by scarring of the cornea, a patient may be offered a corneal transplant.
## Aetiology
Varicella zoster virus (VZV), also known as human herpesvirus-3 (HHV-3)
- previous systemic infection (varicella, i.e. chickenpox)
- virus lies dormant (sometimes for decades) in dorsal root and cranial nerve sensory ganglia
- reactivation leads to herpes zoster (shingles)
- herpes zoster ophthalmicus (HZO) is defined by zoster involvement in the ophthalmic division of the trigeminal nerve
- herpes zoster affects 20-30% of the population at some point in their lifetime; 10-20% of these will develop HZO through involvement of the ophthalmic division of the trigeminal nerve. This represents a lifetime incidence of one in 100 individuals
- most cases of ocular involvement develop within three to four weeks of the initial primary care diagnosis
- vaccination: some countries (e.g. USA, Canada, Australia, Japan, Germany) have a policy of vaccinating children against varicella. There is evidence that this is protective not only against chickenpox but also against herpes zoster in later life. In the UK, such vaccination is offered only to children who are particularly vulnerable to chickenpox, e.g. those undergoing chemotherapy
- in some countries (e.g. USA) herpes zoster vaccination is offered to adults over 60. Public Health England introduced routine herpes zoster vaccination for people aged 70 years in 2013

## Predisposing factors
Age: the peak incidence in healthy individuals is in the 5th to 7th decades, but one in three cases occur in people under the age of 50
Immune compromise: HIV/AIDS, medical immunosuppression

## Symptoms
Pain and altered sensation of the forehead on one side
Rash affecting forehead and upper eyelid appears a day to a week later
General malaise, headache, fever
Ocular symptoms in acute phase
- discomfort
- discharge
- redness
- pain
- photophobia

## Signs
Skin features
- unilateral painful, red, vesicular rash on the forehead and upper eyelid, progressing to crusting after 2-3 weeks; resolution often involves scarring
- periorbital oedema (may close the eyelids and spread to opposite side)
- lymphadenopathy (swollen regional lymph nodes)
- lesion at the side of the tip of the nose (Hutchinson’s sign) indicates three times the usual risk of ocular complications, but these may also occur in one in three patients without the sign

### Ocular lesions
(variable in scope and severity, may be chronic or recurrent)
- mucopurulent conjunctivitis (common), associated with vesicles

---

Herpes Zoster Ophthalmicus (HZO)
Version 14, Page 1 of 4
Date of search 26.02.19; Date of revision 29.03.19; Date of publication ab.cd.ef; Date for review 25.02.21
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Herpes Zoster Ophthalmicus (HZO)

| Differential diagnosis | Ocular lesions: herpes simplex keratitis  
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cutaneous lesions: cellulitis, contact dermatitis, atopic eczema, impetigo</td>
</tr>
</tbody>
</table>

**Management by Optometrist**

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

**Non pharmacological**

Rest and general supportive measures (reassurance, support at home, good diet, plenty of fluids)
Advise avoidance of contact with elderly or pregnant individuals, also babies and children not previously exposed to VZV (who are non-immune) or immunodeficient patients

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

**Pharmacological**

Topical lubricants for relief of ocular symptoms
Pain relief: aspirin, paracetamol or ibuprofen (check history for contraindications). Stronger analgesics (e.g. opiates) may be indicated (co-manage with GP)

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

**Management Category**

**A1:** for acute skin lesions: emergency referral (same day) to GP for systemic anti-viral treatment
Early treatment with oral aciclovir (within 72 hours after rash onset) reduces the percentage of eye disorders in ophthalmic zoster patients from 50% to 20-30%. This early treatment also lessens acute pain

**A3:** first aid measures and urgent referral (within one week) to ophthalmologist if:
- deeper cornea involved
- untreated disciform keratitis can lead to scarring

- keratitis (more than half of all cases)
  - punctate epithelial – early sign, within 2 days (50% of cases)
  - pseudodendrites – fine, multiple stellate lesions (around 4-6 days)
  - nummular – fine granular deposits under Bowman’s layer
  - disciform – 3 weeks after the rash (occurs in 5% of cases)
  - reduced corneal sensation (neurotrophic keratitis)
  - endothelial changes and KP
- episcleritis: occurs in around one third of cases
- scleritis: less common; usually develops after 1 week
- anterior uveitis
- secondary glaucoma
- rarely, posterior segment involvement: retinitis, acute retinal necrosis, choroiditis, optic neuritis, optic atrophy
- rarely, neurological complications: cranial nerve palsy, encephalitis
- post-herpetic neuralgia is defined as pain and/or itch lasting beyond 90 days after the onset of zoster. This affects around 25% of patients and is chronic and severe in about 7%

Complications can occur months or years after the acute phase
o neurotrophic ulceration can lead to perforation
  • anterior uveitis present
  • IOP raised

B3: management to resolution if co-managed with GP and keratitis mild and limited to epithelium
Requires careful monitoring. Maintain low threshold for referral since HZO is associated with chronic and recurrent complications that may be sight threatening

Possible management by Ophthalmologist
- Systemic anti-virals e.g. aciclovir, famciclovir, valaciclovir
- Topical anti-virals (off-licence use)
- Topical steroids
- Immunosuppressive therapy for scleritis
- Botulinum toxin-induced ptosis or surgical tarsorrhaphy for neurotrophic corneal ulceration
- Treat other ocular complications

Evidence base
*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence
Civen R et al. The incidence and clinical characteristics of herpes zoster among children and adolescents after implementation of varicella vaccination. Ped Infect Dis J 2009;28:954-9

Cohen EJ. Management and prevention of herpes zoster ocular disease. Cornea. 2015;34 Suppl 10:S3-8


McDonald EM, de Kock J, Ram FS. Antivirals for management of herpes zoster including ophthalmicus: a systematic review of high-quality randomized controlled trials. Antivir Ther. 2012;17(2):255-64


LAY SUMMARY

Herpes Zoster Ophthalmicus (HZO) is a viral infection of the nerve that supplies sensation (touch and pain) to the eye surface, eyelids, forehead and nose (trigeminal nerve). The virus that affects it (Varicella Zoster Virus [VZV]) also causes chickenpox. Patients who develop HZO, like most people, have usually been exposed to chickenpox by the age of 16 and though they recover from that infection, the virus lies dormant in parts of the brain and spinal cord, with its activity suppressed by the body’s immune system. If, for some reason, that suppression weakens, viruses can become reactivated and travel down the trigeminal nerve, reaching the tissues that it supplies and causing inflammation. When the skin is involved, the condition is known as shingles. Shingles occurs more often and is likely to be more severe in older people whose immunity to VZV is weaker, and in people whose immune system is not functioning normally, as for example in HIV/AIDS, or is suppressed by medical treatment.

In HZO the skin of one side of the forehead and scalp is affected, along with the eye on the same side. Any part of the eye can be involved, but most commonly it is the eye surface, including the conjunctiva (the white of the eye) and the cornea (the clear window of the eye). The cornea reacts in various ways; the most serious long-term effects result from damage to the corneal nerves, causing loss of sensation.

When HZO first appears, patients benefit from anti-viral tablets prescribed as soon as possible, usually by the GP. Mild cases can be co-managed by the optometrist and the GP but more severe cases need to be referred to the ophthalmologist.

Public Health England has introduced shingles vaccination for certain people aged between 70 and 80. This is given once and provides a good measure of protection against the condition.
# Hordeolum

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>There are two types:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- external hordeolum (stye) – acute bacterial infection of the lash follicle and its associated gland of Zeis or Moll</td>
</tr>
<tr>
<td></td>
<td>- internal hordeolum – acute bacterial infection of Meibomian gland</td>
</tr>
<tr>
<td></td>
<td>These infections are usually staphylococcal</td>
</tr>
</tbody>
</table>

| Predisposing factors | Chronic blepharitis |

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Tender lump in eyelid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Epiphora</td>
</tr>
<tr>
<td></td>
<td>Local redness of eye and lid</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs</th>
<th><strong>External hordeolum</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tender inflamed swelling of the lid margin. May point anteriorly through the skin</td>
</tr>
<tr>
<td></td>
<td>Occasionally, multiple abscesses involve entire eyelid</td>
</tr>
<tr>
<td></td>
<td><strong>Internal hordeolum</strong></td>
</tr>
<tr>
<td></td>
<td>Tender inflamed swelling within the tarsal plate. More painful than a stye. May point anteriorly through the skin or posteriorly through the conjunctiva</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differential diagnosis</th>
<th>Preseptal cellulitis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Haematoma of eyelid</td>
</tr>
<tr>
<td></td>
<td>Acute dacryocystitis</td>
</tr>
<tr>
<td></td>
<td>Chalazion (blockage of Meibomian gland with cyst formation)</td>
</tr>
<tr>
<td></td>
<td>Sebaceous cell carcinoma</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Management by Optometrist</th>
<th>Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non pharmacological</td>
<td>Most resolve spontaneously or discharge, followed by resolution</td>
</tr>
<tr>
<td></td>
<td>In the case of external hordeola, it may help to remove the lash associated with the infected follicle</td>
</tr>
<tr>
<td></td>
<td>Traditional remedies such as hot spoon bathing and/or warm compresses may relieve symptoms</td>
</tr>
<tr>
<td></td>
<td>Manage associated blepharitis with lid hygiene (see Clinical Management Guideline on Blepharitis)</td>
</tr>
<tr>
<td></td>
<td>(GRADE*: Level of evidence=low; Strength of recommendation=strong)</td>
</tr>
<tr>
<td></td>
<td>Rarely, refer for incision in cases that do not discharge (commoner with internal hordeolum)</td>
</tr>
<tr>
<td></td>
<td>An internal hordeolum may evolve into a chalazion (see Clinical Management Guideline on Chalazion)</td>
</tr>
<tr>
<td></td>
<td>Advise patient to return/seek further help if symptoms persist</td>
</tr>
</tbody>
</table>

| Pharmacological           | Consider course of antibiotic drops or ointment (e.g. chloramphenicol) in the presence of copious muco-purulent discharge |
|                          | In severe or recurrent cases, consider management with a systemic broad-spectrum antibiotic |
|                          | (GRADE*: Level of evidence=low; Strength of recommendation=strong) |

<table>
<thead>
<tr>
<th>Management Category</th>
<th>B2: Alleviation/palliation: normally no referral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible management by Ophthalmologist</td>
<td>Possible incision, but surgery rarely performed in presence of acute</td>
</tr>
</tbody>
</table>
**Hordeolum**

<table>
<thead>
<tr>
<th>Evidence base</th>
</tr>
</thead>
<tbody>
<tr>
<td>*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see <a href="http://www.gradeworkinggroup.org/index.htm">http://www.gradeworkinggroup.org/index.htm</a>)</td>
</tr>
</tbody>
</table>

**Sources of evidence**


**LAY SUMMARY**

A hordeolum is an acute bacterial infection of the glands of the eyelid. An internal hordeolum affects the Meibomian (oil) glands within the eyelids whereas an external hordeolum (commonly referred to as a stye) affects the glands associated with the eyelashes. Both conditions cause red and tender swellings of the eyelid. Traditional remedies such as hot spoon bathing and/or warm compresses may relieve symptoms. In some cases, treatment with antibiotic drops or ointment is needed to speed up resolution. In the case of a severe infection, antibiotic tablets may be required.
**Keratitis, CL-associated infiltrative**

| Aetiology | Contact lens-associated infiltrative events, including:  
|           | • contact lens-associated peripheral ulcer (CLPU)  
|           | • contact lens-associated infiltrative keratitis  
|           | The aetiology of this condition is inflammatory, not infective. Though it is bacteria-related, bacteria do not invade or replicate in the cornea and there is no progression to infection, nor is the condition a marker for increased risk of microbial keratitis, which is a separate disease entity  
|           | CL-associated infiltrative keratitis is considered to be a response to microbial (usually *Staphylococcal*) antigens, derived from bacteria on the lens or on the lid margin. Micro-organisms cannot usually be recovered from the lesions  
|           | Incidence of contact lens associated inflammatory events in daily disposable silicon hydrogel lenses has been reported as 0.4% per year of wear; incidence is higher in re-usable lenses and much higher in extended wear  

| Predisposing factors | Bacterial bioburden of eyelid margins, contact lenses and contact lens cases  
|                      | Multipurpose contact lens solutions  
|                      | Poor hand hygiene  
|                      | Smoking  

| Symptoms | Eye moderately red and slightly watery  
|          | Mild foreign body sensation  
|          | Mild photophobia  
|          | (NB: symptoms vary in severity; some cases are asymptomatic)  

| Signs | Peripheral anterior stromal infiltrate, single or multiple  
|      | Usually small (generally less than 1.0mm in diameter)  
|      | Overlying epithelium usually stains with fluorescein  
|      | Conjunctival hyperaemia, mild  
|      | Epiphora, mild (or absent)  
|      | Anterior chamber quiet or mildly inflamed  
|      | No lid oedema  
|      | Usually unilateral  

| Differential diagnosis | Microbial (bacterial or fungal) keratitis  
|                       | • appearance can be similar, therefore monitor closely especially over the first 24 hours and if diagnosis remains in doubt, refer to ophthalmologist as an emergency  
|                       | Marginal keratitis  
|                       | Corneal scar  
|                       | Herpes simplex keratitis  
|                       | Adenovirus keratoconjunctivitis  

**Management by Optometrist**

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

| Non pharmacological | Temporarily discontinue lens wear  
|                     | • most signs and symptoms resolve within 48 hours  

Keratitis, CL-associated infiltrative  
Version 5, Page 1 of 3  
Date of search 20.05.17; Date of revision 20.09.17; Date of publication 05.12.17; Date for review 19.05.19  
© College of Optometrists
### Keratitis, CL-associated infiltrative

<table>
<thead>
<tr>
<th>Management Category</th>
<th>B2: alleviation / palliation: normally no referral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible management by Ophthalmologist</td>
<td>Not normally referred</td>
</tr>
</tbody>
</table>

**Pharmacological**
- Ocular lubricants for symptomatic relief
- Oral antibiotic (tetracycline group) may be indicated for blepharitis (see Clinical Management Guideline on Blepharitis)

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

**Sources of evidence**

- Szczotka-Flynn L, Jiang Y, Raghupathy S, Bielefeld RA, Garvey MT, Jacobs MR, Kern J, Debane SM. Corneal inflammatory events with daily silicone hydrogel lens wear. Optom Vis Sci. 2014;91:3-12

**LAY SUMMARY**

This condition, affecting contact lens wearers, has been given many different names. It is an inflammation of the cornea (the clear window of the eye) caused by a reaction to bacteria on the surface of the contact lens. Patients experience slight discomfort, redness and watering of the eye (it usually affects just one eye) and they may be unduly sensitive to light. The optometrist will see a small opaque area or areas near the edge of the cornea, plus inflammation of the white of the eye.
Keratitis, CL-associated infiltrative

The most important task of the optometrist is to distinguish between this condition and an actual infection of the cornea, which is a sight-threatening emergency. Stopping contact lens wear usually allows the symptoms and most of the signs to resolve within 48 hours.

Patients will usually need to be reminded of contact lens hygiene measures, including hand washing before handling their lenses, the need to replace their lens case frequently and avoiding overnight wear of their lenses.
Aetiology | Toxic or hypersensitivity response to bacterial (e.g. *Staphylococcal*) exotoxins
---|---
Predisposing factors | Bacterial (e.g. *Staphylococcal*) blepharitis
| Current or recent upper respiratory tract infection
| Condition tends to be recurrent
Symptoms | Ocular discomfort increasing to pain
| Lacrimation
| Red eye
| Photophobia
Signs | Ulcer (stromal infiltrate with overlying epithelial loss) which may be round or arcuate, single or multiple, unilateral or bilateral, adjacent to limbus, and separated from limbus by interval of clear cornea
| Ulcer stains with fluorescein
| Hyperaemia and oedema of adjacent bulbar conjunctiva
Differential diagnosis | Other causes of ulceration of the peripheral cornea:
| • microbial keratitis
| • contact lens-associated corneal infiltrate
| • rosacea keratitis
| • Mooren’s ulcer
| • peripheral keratitis associated with rheumatoid arthritis or other systemic collagen vascular disease
| • corneal phlyctenulosis
| • Terrien’s marginal degeneration
| • marginal herpes simplex keratitis

**Management by Optometrist**

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.

Non pharmacological | Sunglasses to ease photophobia
| (GRADE*: Level of evidence=low, Strength of recommendation=strong)
Pharmacological | Ocular lubricants for symptomatic relief (drops for use during the day, unmedicated ointment for use at bedtime)
| Systemic analgesia if needed: paracetamol, aspirin or ibuprofen
| Regular lid hygiene for associated blepharitis (with a view to limiting recurrence)
| (GRADE*: Level of evidence=low, Strength of recommendation=strong)

Marginal keratitis is a self-limiting condition. Nevertheless it is conventional to give pharmacological treatment with a view to relieving symptoms and shortening the clinical course. The concurrent use of topical antibiotic (e.g. chloramphenicol) to reduce bacterial load, in addition to topical steroid (e.g. prednisolone) to reduce inflammation, is theoretically justified. However, the immunosuppressive effect of the steroid enhances the risk of infection
| (GRADE*: Level of evidence=low, Strength of recommendation=weak)

**Management Category** | **B3**: Management to resolution
If persistent or recurrent, refer to ophthalmologist

**Possible management by Ophthalmologist**

Microbiological cultures of lesion and lid margins
Investigation of patient’s immune status
Topical and/or systemic antibiotic treatment of blepharitis

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and
LAY SUMMARY
This is a slightly unusual condition caused by a reaction to the presence of bacteria near the eye, for example on the edges of the eyelids. It is an inflammation, not an infection. Patients experience redness, watering and pain in the eye. A shallow ulcer develops at the edge of the cornea (the clear window of the eye), which can resemble a number of other conditions including infection. The condition usually resolves by itself, but it may be dealt with more quickly if steroid and antibiotic drops are prescribed. If blepharitis (inflammation of the edges of the eyelids) is the cause, this should be treated.
## Photokeratitis (Ultraviolet [UV] burn, Arc eye, Snow Blindness)

### Aetiology

<table>
<thead>
<tr>
<th>Exposure to UVB (290 to 320nm) or UVC (100 to 290nm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sources: welding arcs, sun (including reflection from snow or water), tanning lamps, therapeutic high intensity UV (for skin conditions or seasonal affective disorder), germicidal UV lamps, other sources of UVB or UVC</td>
</tr>
<tr>
<td>Absorption of radiation by corneal epithelium causing punctate erosions</td>
</tr>
</tbody>
</table>

### Predisposing factors

Lack of suitable eye protection

### Symptoms

<table>
<thead>
<tr>
<th>Delay of 6-12 hours between exposure and onset of symptoms is usual; however, latency varies inversely with exposure dose and can be as short as 1 hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild cases: irritation and foreign body sensation</td>
</tr>
<tr>
<td>Severe cases: pain, redness, photophobia, blepharospasm, lacrimation, blurring of vision</td>
</tr>
</tbody>
</table>

### Signs

<table>
<thead>
<tr>
<th>Bilateral (if unilateral, suspect corneal or subtarsal foreign body)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lid chemosis and redness</td>
</tr>
<tr>
<td>Conjunctival hyperaemia</td>
</tr>
<tr>
<td>Epiphora</td>
</tr>
<tr>
<td>Punctate staining of corneal epithelium with fluorescein (may be coalescent)</td>
</tr>
<tr>
<td>Mild transitory visual loss</td>
</tr>
<tr>
<td>Associated skin burns from UV exposure</td>
</tr>
</tbody>
</table>

### Differential diagnosis

<table>
<thead>
<tr>
<th>Acute viral keratoconjunctivitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contact or toxic keratitis</td>
</tr>
<tr>
<td>Contact lens overwear</td>
</tr>
<tr>
<td>Dry eye</td>
</tr>
<tr>
<td>Foreign body</td>
</tr>
</tbody>
</table>

### Management by Optometrist

<table>
<thead>
<tr>
<th>Non pharmacological</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reassure patient that damage is transitory</td>
</tr>
<tr>
<td>symptoms will be gone within 24 to 48 hours (mild photophobia and blurring may persist for a week or longer)</td>
</tr>
<tr>
<td>Cold compresses, sunglasses for symptomatic relief</td>
</tr>
<tr>
<td>Advise rest with eyes closed</td>
</tr>
<tr>
<td>Review following day (corneal epithelium should have largely healed)</td>
</tr>
<tr>
<td>Advise patient to return/seek further help if symptoms persist</td>
</tr>
<tr>
<td>Advise patient on future eye protection (GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pharmacological</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local anaesthetic (benoxinate or proxymetacaine) should be used only if required to aid examination, and not for pain relief</td>
</tr>
<tr>
<td>Drops: tear supplements (preferably unpreserved) for symptomatic relief</td>
</tr>
<tr>
<td>Ointment: unmedicated (to ease discomfort through lubrication)</td>
</tr>
<tr>
<td>Oral analgesic for pain relief (GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>
**Photokeratitis (Ultraviolet [UV] burn, Arc eye, Snow Blindness)**

<table>
<thead>
<tr>
<th>Management Category</th>
<th>B3: Management to resolution by Optometrist</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible management by Ophthalmologist</td>
<td>Not normally required</td>
</tr>
</tbody>
</table>

| Antibiotic (e.g. gutt. chloramphenicol) as prophylaxis against infection, if risk high  |
| Cycloplegic (short acting: e.g. gutt. cyclopentolate 1%) to relieve pain of ciliary spasm |
| (GRADE*: Level of evidence=low, Strength of recommendation=weak) |

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

**Sources of evidence**

- Cullen AP. Photokeratitis and other phototoxic effects on the cornea and conjunctiva. Int J Toxicol. 2002;21:455-64

**LAY SUMMARY**

This condition is also known as Arc Eye and Snow Blindness. It is caused when the eyes are exposed to too much ultraviolet (UV) light. After a delay of 6 to 12 hours following exposure to arc welding, sun lamps or other sources of UV light, the eyes become red, painful, watery and unduly sensitive to light. The vision may become blurred and the eyelids may be red and swollen. These symptoms are caused by temporary damage to the cells on the surface of the eye. Fortunately the condition gets better by itself and there is usually no permanent damage. Eye drops can be prescribed or purchased to make the eyes more comfortable while they recover. Pain relief tablets may be needed also.
Microbial keratitis (Acanthamoeba sp.)

**Aetiology**

*Acanthamoebae* are ubiquitous free-living protozoans, present in:
- well water, drains, soil, dust
- often present in domestic tap water (especially from storage tanks)

Can exist in two forms
- motile, feeding and replicating form: trophozoite (most common form found in water and easily destroyed)
- dormant form: cyst (highly resistant to disinfection, can survive for long periods in hostile environments)

*Acanthamoeba* keratitis is rare in the general population (estimated annual incidence: 1.4 per million per annum) but much commoner in contact lens wearers.

In the UK, higher incidence in hard water districts and where bathrooms are supplied by tank-stored water.

In 10% of cases there is associated scleritis. *Acanthamoeba* sclerokeratitis is associated with a poor clinical outcome.

**Predisposing factors**

Contact lens wear is associated with >90% cases of *Acanthamoeba* keratitis
- majority are soft lenses (particularly reusable or extended wear)
- inadequate disinfection
- use of non-sterile solutions
- tap water rinsing of lenses and/or storage cases
- contamination of storage case with bacteria and fungi (± biofilm) which provide substrate for *Acanthamoebae*
- exposure to shower, pool, or hot tub water

Agricultural injuries

**Symptoms**

- pain (may be severe and out of proportion to degree of ocular inflammation; may also be painless in the early stages)
- visual loss
- redness
- epiphora
- photophobia

Can be bilateral

May be a long history and condition may be misdiagnosed as herpetic, bacterial or fungal keratitis

*NB in earliest stages, pain may be minimal*

**Signs**

**Early signs**
- epithelial or subepithelial infiltrates
- pseudodendrites
- radial keratoneuritis (infiltrates along corneal nerves)
- recurrent breakdown of the corneal epithelium

**Later signs**
- deep inflammation of the cornea consisting of a central or paracentral ring-shaped or disciform infiltrate or abscess
- stromal thinning
- extension of inflammation into sclera
- anterior chamber cells and flare
- hypopyon

**Differential diagnosis**

Signs may masquerade as herpes simplex with temporary improvement on anti-herpetic treatment, further delaying diagnosis

Suspect any painful epitheliopathy that:
### Microbial keratitis (Acanthamoeba sp.)

- does not respond to normal treatment
- has known risk factor (e.g. contact lens wear or corneal trauma associated with soil or contaminated water)

*(NB: Dendritic keratitis in a contact lens wearer should raise the index of suspicion of an Acanthamoeba infection)*

Bacterial or fungal keratitis
Concurrent bacterial and/or fungal infection can occur

### Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

<table>
<thead>
<tr>
<th>Non pharmacological</th>
<th>Cease contact lens wear immediately (both eyes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharmacological</td>
<td>None</td>
</tr>
</tbody>
</table>

**Management Category**

- A1: emergency (same day) referral to ophthalmologist without intervention. *Acanthamoeba* keratitis can be difficult to treat; therefore prompt, aggressive therapy is vital. Delay is associated with a poorer prognosis. Telephone the on-call ophthalmologist. Advise patient to take lenses and lens case for possible culture

### Possible management by Ophthalmologist

**Diagnosis** will usually be confirmed by histology (corneal scrape) and culture or PCR. Cystic form can also be imaged by confocal microscopy

Intensive (day and night) topical medical treatment with either a biguanide or a diamidine or a combination of the two:

- Biguanides: polyhexamethylene biguanide (PHMB), chlorhexidine
- Diamidines: propamidine (Brolene), dibromopropamidine (Brolene ointment), hexamidine

Continuous treatment may be necessary for weeks or months

*(A recent systematic review identified a paucity of evidence to inform robust conclusions for treating AK in practice)*

- Systemic analgesia as necessary
- Topical steroid to limit inflammation
- Topical antibiotics as necessary for secondary bacterial infection
- Penetrating keratoplasty if corneal irregularity, thinning and/or scarring is severe following complete control of infection

### Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation* (see http://gradeworkinggroup.org/toolbox/index.htm)

**Sources of evidence**

Microbial keratitis (Acanthamoeba sp.)


LAY SUMMARY

Acanthamoeba is a protozoan (single-celled organism) that is very widespread throughout the environment, especially where there is standing water, and it may be present in inadequately treated tap water. It has two forms: the trophozoite, which is active, capable of feeding, moving and reproducing, and which is easily destroyed; and the cyst, which is dormant and difficult to destroy. Acanthamoeba can change between these two forms, depending on whether it is in a favourable or a hostile environment.

Acanthamoeba is normally harmless to humans, but if it is transferred to the eye on a contaminated contact lens it can infect the cornea (the clear window at the front of the eye). Such infections can be difficult to treat owing to the lack or non-availability of anti-amoebic drugs. It is far better to prevent the infection by the use of effective contact lens hygiene, in particular avoiding contact of the lens and lens case with tap water.

Patients with early Acanthamoeba keratitis usually complain of discomfort, redness and light sensitivity of the affected eye. In the later stages the eye can become very painful as the nerves and deeper parts of the cornea become affected.

An optometrist who suspects such an infection is advised to refer the patient as an emergency (same day) to the ophthalmologist, who will try to confirm the diagnosis and then prescribe special eye drops given day and night. Often the patient will be admitted to hospital. If there is much scarring of the cornea following the eventual elimination of the infection, and vision is badly affected, a corneal transplant may be recommended.
## Aetiology

The commonest bacterial corneal pathogens are:
- *Pseudomonas* sp. (Gram -ve)
- *Staphylococcus* sp. (Gram +ve)
- *Streptococcus* sp. (Gram +ve)
- other Gram -ve organisms

Note: severe contact lens-related infections tend to be Gram –ve, particularly *Pseudomonas* species

Fungal keratitis is rare in the UK but common in some other parts of the world. The most common fungal corneal pathogens are:
- *Candida* sp. (yeast-like)
- *Fusarium* sp. (filamentous)
- *Aspergillus* sp (filamentous)

## Predisposing factors

Bacterial keratitis is usually associated with one or more of the following:
- contact lens wear, especially soft lenses worn overnight (incidence soft daily wear: 2-4 per 10,000 per year, soft overnight wear: 20 per 10,000 per year). The anticipated reduction in contact lens-related microbial keratitis with silicone hydrogel lenses has not been observed. Other main risk factors for CL-related MK are:
  - increased days of wear
  - poor hand, lens and storage case hygiene
  - youth
  - male gender
  - smoking
  - internet purchase of lenses, particularly cosmetic lenses
- ocular surface disease, including:
  - corneal exposure
  - corneal decompensation
  - chronic epithelial defect
  - neurotrophic keratopathy, e.g. secondary to HSK or diabetes
- ocular trauma or surgery, including loose or broken sutures
- immune compromise
- topical steroid use
- lid margin infection (usually *Staphylococcal*)

Fungal keratitis (filamentous) is usually secondary to trauma involving organic material; it can also be contact lens or solution related. Fungal keratitis (yeast-like) most usually complicates ocular surface disease or in immunocompromised patients

## Symptoms

Pain, moderate to severe (usually acute onset, rapid progression)
Redness, photophobia (may be severe), discharge, blurred vision (especially if lesion on visual axis)
Awareness of white or yellow spot on cornea
Usually unilateral

## Signs

Lid oedema
Epiphora
Microbial keratitis (bacterial, fungal)

<table>
<thead>
<tr>
<th>Discharge</th>
<th>(mucopurulent or purulent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conjunctival hyperaemia and infiltration</td>
<td></td>
</tr>
<tr>
<td>Corneal lesion usually single (central or mid-peripheral)</td>
<td></td>
</tr>
<tr>
<td>• excavation of epithelium, Bowman's layer, stroma (tissue necrosis)</td>
<td></td>
</tr>
<tr>
<td>• stromal infiltration beneath lesion</td>
<td></td>
</tr>
<tr>
<td>• stromal oedema with folds in Descemet's membrane</td>
<td></td>
</tr>
<tr>
<td>• endothelial fibrin plaque beneath lesion</td>
<td></td>
</tr>
<tr>
<td>Anterior chamber activity (flare, cells, hypopyon or coagulum if severe)</td>
<td></td>
</tr>
</tbody>
</table>

Fungal keratitis produces similar signs to bacterial keratitis, though the infection may develop more slowly (however *Fusarium* infection can progress rapidly and invasively)

<table>
<thead>
<tr>
<th>Differential diagnosis</th>
<th>Corneal infiltrative lesions (contact lens related or marginal keratitis; see separate Clinical Management Guidelines)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• peripheral, small (0.5-1.5 mm) with less anterior chamber response</td>
</tr>
<tr>
<td></td>
<td>• not a marker for increased risk of bacterial infection</td>
</tr>
</tbody>
</table>

*Acanthamoeba* keratitis (see Clinical Management Guideline)

<table>
<thead>
<tr>
<th>Management by Optometrist</th>
<th>Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non pharmacological</td>
<td>Warn contact lens wearers not to discard their lenses or lens cases, but to retain them for culture (GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>Usually none. Beginning empirical antimicrobial therapy without laboratory evaluation may delay correct diagnosis and proper care if improvement does not promptly take place (GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

| Management Category                    | A1: emergency referral to an ophthalmologist; no intervention. Severe sight-threatening condition. Telephone on-call ophthalmologist. |

**Possible management by Ophthalmologist**

<table>
<thead>
<tr>
<th>Corneal scrape (for culture and determination of antibiotic sensitivities) followed by initiation of intensive (round the clock) treatment with one or more antibiotics. Corneal isolate studies show changing patterns of bacterial pathogenesis and the development of resistant strains.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• monotherapy: fluoroquinolones (e.g. levofloxacin, moxifloxacin) are adequate for most cases but not for resistant species of <em>Staphylococcus aureus</em> and <em>Pseudomonas aeruginosa</em></td>
<td></td>
</tr>
<tr>
<td>• dual therapy: the recommended fortified agents (a cephalosporin and an aminoglycoside) are not commercially available</td>
<td></td>
</tr>
<tr>
<td>• may be combined with systemic antibiotics if lesion close to limbus</td>
<td></td>
</tr>
</tbody>
</table>

Polymerase chain reaction [PCR] technique may be used to identify causative organisms.
Possible admission to hospital when good compliance is unlikely, or for overnight treatment of severe infections (axial lesions, lesions 6mm or more in diameter, or with 50% or more stromal thinning)

Cycloplegia

Hypotensive agents for secondary glaucoma

Topical steroids (only when infection controlled) – not well supported by evidence base

Amphotericin B (as 0.15% eye drops) is the drug of choice in fungal keratitis caused by yeasts (e.g. Candida)

Fungal infections sometimes require combined topical (e.g. natamycin 5%, econazole 1% or voriconazole 1%) and oral (e.g. voriconazole) therapy. Clinical strategies continue to evolve

Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence


FlorCruz NV, Evans JR. Medical interventions for fungal keratitis. Cochrane Database Syst Rev. 2015;4:CD004241


LAY SUMMARY

This is a serious condition in which the cornea (the clear window of the eye) becomes infected. The usual cause is contact lens wear, but infection of the cornea can also result from abnormal exposure of the eye, for example if the eyelids are not blinking normally, loss of sensation in the cornea, or from medical devices such as contact lenses.
Microbial keratitis (bacterial, fungal)

eye surface, injury or surgery, lack of tears (dry eye), and in people whose immune system is not functioning properly. The usual cause is bacterial (i.e. caused by a common germ) but some cases are due to fungal infection.

Microbial keratitis is a very serious condition. It usually begins suddenly with redness and pain in one eye. The eye waters and there may be a discharge. Light may hurt the eye, making it difficult to open. The vision of the eye may be blurred.

The optometrist will usually observe an area in the cornea where the clear tissue has been turned cloudy by infection. There may be an ulcer on the surface. The inflammation extends into the chamber at the front of the eye also.

Microbial keratitis is a sight-threatening emergency, so such patients should be referred immediately to the ophthalmologist. The ophthalmologist will take specimens from the ulcer and begin treatment with antibiotic eye drops. These will need to be put into the eye very frequently. The patient will most likely be admitted to hospital so that treatment can continue day and night. If the infection is caused by a fungus, the treatment will usually be with anti-fungal eye drops and sometimes with tablets.

When the infection is controlled, steroid eye drops may be added. If the infection was contact lens related, the patient will be given advice on whether it is safe to wear lenses again.
Aetiology
- Localised dermal infection caused by a poxvirus
- Commonest in:
  - children up to the age of 14 years, with the highest incidence in the age range 1-4 years in the UK
  - immuno-compromised adults (e.g. HIV +ve)
- Transmitted by skin-to-skin contact; mildly contagious
- Lesions on the lid margins may shed viral toxins into the conjunctival sac, causing
  - follicular conjunctivitis
  - pannus

Predisposing factors
- Epidemiological studies have shown separate associations with attendance at swimming pools, and with eczema

Symptoms
- Presence of skin lesion(s)
- Ocular symptoms: redness, watering, photophobia, blurring of vision (all mild)

Signs
- Skin nodule(s) (typically 2-3 mm diam), often with a central depression (‘umbilicated’)
- No visible inflammation
- Central core has cheese-like or waxy material which may discharge spontaneously
- May be single or multiple on the lid(s) and/or elsewhere on the body
- Ocular signs (usually unilateral)
  - hyperaemic conjunctiva
  - conjunctival follicles
  - pannus (in long-standing cases)
  - watery discharge
- No lymphadenopathy

Differential diagnosis
- Other lesions of the lids which may be skin-coloured:
  - basal cell carcinoma, neurofibroma, sebaceous adenoma, non-pigmented intradermal naevus, squamous cell papilloma, chalazion, cutaneous horn, sebaceous carcinoma
- Other causes of follicular conjunctivitis:
  - viral: herpes simplex, adenovirus, chicken pox lesions around eye
  - chlamydia
  - topical medication (conjunctivitis medicamentosa)
  - Parinaud’s oculo-glandular syndrome

Management by Optometrist
- Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

Non pharmacological
- Usually self-limiting (weeks or months) without scarring or other long term sequelae. Although no reliable evidence-based recommendations can be given for the treatment of molluscum contagiosum at present, clinical consensus would support active intervention for lesions of the lid margin causing follicular conjunctivitis (GRADE*: Level of evidence=low, Strength of recommendation=strong)

- If lesion is quiet (dry central core) and no ocular symptoms or associated follicular conjunctivitis:
Molluscum contagiosum

**Lay Summary**

Molluscum contagiosum is a viral infection of the skin that occurs commonly in children. It usually consists of a single or a small group of fluid-filled pimples which have an obvious central depression. These can occur on the eyelids or surrounding skin. If molluscum occurs close to the eye, it may cause a type of conjunctivitis. Since the condition gets better by itself, the usual advice to patients with this condition is to monitor without treatment to allow natural healing. However, molluscum removal may be recommended if there are several pimples near the eye, if it occurs on the rim of the eyelid, or when conjunctivitis is present. Various surgical and medical treatments are available but there is no evidence that one type of treatment is better than another.

**Management Category**

<table>
<thead>
<tr>
<th>B2: Alleviation / palliation: normally no referral</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1: Routine referral to ophthalmologist if:</td>
</tr>
<tr>
<td>• multiple peri-ocular lesions</td>
</tr>
<tr>
<td>• lesions on the lid margin</td>
</tr>
<tr>
<td>• follicular conjunctivitis</td>
</tr>
</tbody>
</table>

**Possible management by Ophthalmologist**

Possible destruction of lesion by shave excision, cautery, cryotherapy or incision and curettage

**Pharmacological**

Artificial tears and lubricating ointment may relieve symptoms in follicular conjunctivitis

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*

**Sources of evidence**


**Lay Summary**

Molluscum contagiosum is a viral infection of the skin that occurs commonly in children. It usually consists of a single or a small group of fluid-filled pimples which have an obvious central depression. These can occur on the eyelids or surrounding skin. If molluscum occurs close to the eye, it may cause a type of conjunctivitis. Since the condition gets better by itself, the usual advice to patients with this condition is to monitor without treatment to allow natural healing. However, molluscum removal may be recommended if there are several pimples near the eye, if it occurs on the rim of the eyelid, or when conjunctivitis is present. Various surgical and medical treatments are available but there is no evidence that one type of treatment is better than another.
### Aetiology

**Congenital**
Between 5% and 20% of babies have a persistent membranous obstruction at opening of nasolacrimal duct into nose; unilateral or bilateral

**Acquired**
Usually idiopathic (i.e. no established cause)
Usually involutinal (i.e. age-related) stenosis (abnormal narrowing) of nasolacrimal passages

Punctal or canalicular stenosis/occlusion
- post-infective (chlamydia, herpes simplex/zoster, staphylococcus)
- post-cicatricial disease (ocular pemphigoid, Stevens-Johnson)
- post-conjunctivitis medicamentosa (see Clinical Management Guideline on Conjunctivitis medicamentosa)
- iatrogenic (e.g. repeated probing, eyelid surgery)
- secondary to ectropion or punctal eversion

Mechanical (trauma, tumours, obstruction by lashes or foreign matter, e.g. dacryoliths [calcium stones], punctal/canalicular plugs)

Infection of canaliculi (canaliculitis)
- rarely, due to Actinomyces (*Streptothrix* sp.) – a Gram-positive bacillus
- such cases usually unilateral
- ‘pouting’ punctum typical

Local infection (chronic sinusitis, dacryocystitis)

### Predisposing factors

**Age:** *congenital* in babies, *acquired* in later life
**Female gender**

One case series identified a history of infective conjunctivitis and regular swimming in chlorinated water as predisposing factors

Other factors: see above

### Symptoms

Epiphora and mucous discharge
Irritation
Blurred vision due to excessive tear meniscus, especially on downgaze, e.g. when reading

### Signs

**Congenital**
Epiphora and mucous discharge; mucopurulent if infected
Pressure over lacrimal sac may cause reflux of mucous material from puncta

**Acquired**
Check puncta for
- size (normally 0.2 to 0.3 mm diameter)
- apposition to the globe and marginal tear strip
- contact with opposite lid on eye closure

### Differential diagnosis

**Congenital**
Congenital glaucoma
Punctal atresia (congenital absence or abnormal narrowing of puncta)

**Acquired**
Rule out inflammation or infection (pain, discharge, swelling, redness, mucus reflux on syringing in adults, history of sinusitis)
- canaliculitis (chronic mucopurulent conjunctivitis, punctum expresses chalky concretions, redness & tenderness over canaliculi)
- dacryocystitis (distended tender lacrimal sac)
Nasolacrimal duct obstruction (nasolacrimal drainage dysfunction)

Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

Non pharmacological

**Congenital**

**Diagnostic test**

Fluorescein disappearance test
- a drop of 1% fluorescein should disappear from the tear meniscus in 5-10 minutes (cobalt blue light, room lights off); any longer suggests partial or complete obstruction

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

**Therapy**

Do not syringe or probe
Instruct parent in massage. Gentle pressure with finger over common canaliculus, stroking downwards firmly to raise pressure in lacrimal sac and encourage opening of valve. Suggest ten strokes, twice daily
Regular cleaning of discharge from lids

High rate of spontaneous resolution during the first 12 months of life (>50% resolve within 6 months, 70% by 12 months). Children with unilateral obstruction may benefit from early probing by ophthalmologist

(GRADE*: Level of evidence=moderate, Strength of recommendation=strong)

**Acquired**

**Diagnostic tests**

Lacrimal syringing (NB not in congenital cases)
- instil a drop of topical anaesthetic
- gently dilate punctum with punctal dilator
- syringe with normal saline via lacrimal cannula
- if saline passes into nose (patient swallows and tastes salt) – nasolacrimal system is patent
- if there is resistance to the passage of the cannula and reflux from opposed canaliculus – common canaliculus is stenosed
- if no saline passes into nose – complete lacrimal duct obstruction

Jones fluorescein dye test
- significant amount of fluorescein remaining in tear meniscus two minutes or more after instillation indicates restricted drainage
- check for appearance of fluorescein in the nose (examine tissue after nose blow; if fluorescein present, lacrimal system is patent)
- place anaesthetic-soaked cotton bud in nose (if bud stained with fluorescein after 5 min, lacrimal system is patent)

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

**Therapy**

Lacrimal lavage (saline syringing) may be effective in some cases:
- local (discrete) obstruction
Nasolacrimal duct obstruction (nasolacrimal drainage dysfunction)

- subacute inflammation or infection
  It is less likely to be effective:
  - in stenosis in the elderly
  - where there is an underlying disease (inflammation, tumour)
  (GRADE*: Level of evidence=low, Strength of recommendation=strong)

**Pharmacological**

**Congenital**
Topical broad spectrum antibiotic e.g. chloramphenicol drops (only if clinical evidence of infection)
(GRADE*: Level of evidence=low, Strength of recommendation=weak)

**Acquired**
Topical broad spectrum antibiotic e.g. chloramphenicol drops (only if clinical evidence of infection)
(GRADE*: Level of evidence=low, Strength of recommendation=weak)

**Management Category**

**Congenital**
- B2: alleviation or palliation; normally no referral (due to high rate of spontaneous resolution during the first 12 months of life; children with unilateral obstruction may benefit from early probing by ophthalmologist)

**Acquired**
- B1: Initial management (including drugs) followed by routine referral

Possible management by Ophthalmologist

- Lacrimal syringing
- Probing (through puncta, canaliculi, sac, to nasolacrimal duct)
  - Congenital: usually not until 12 months of age to allow for spontaneous canalisation
  - in resistant acquired cases has limited success and carries risk of aggravation of underlying disease and of tissue trauma
- Canalicular curettage
  - for Actinomyces infection
- Punctal dilatation where there is stenosis
- Surgical removal of posterior wall of vertical limb of canaliculus
  - considered when repeated punctal dilatation ineffective
- X-ray imaging of radiopaque liquid injected into the lacrimal drainage system (dacryocystogram, DCG)
  - pinpoints any obstructions and guides surgery
- Surgery in canalicular or nasolacrimal duct obstruction includes
  - dacryocystorhinostomy, DCR (surgical or endolaser)
  - if other measures have failed, insertion of a Lester-Jones tube

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence


Lin AE, Chang YC, Lin MY, Tam KW, Shen YD. Comparison of treatment for congenital nasolacrimal duct obstruction: a systematic review and
Nasolacrimal duct obstruction (nasolacrimal drainage dysfunction)

|---|

LAY SUMMARY

The narrow tube that collects tears from the inner corner of the each eye and drains them to the inside of the nose is called the nasolacrimal duct. Normally it functions well but it may sometimes become blocked, causing the eye to water. This may happen as the result of infection, inflammation or the effects of medications or surgery. Occasionally babies are born with the duct(s) not fully open, but most of these go on to open without treatment before the age of 12 months.

In adults, the blockage can be confirmed by attempting to pass a little saline solution through the nasolacrimal duct, using a small syringe. Fluorescein dye can also be used to test whether the duct is free. However, syringing by itself rarely cures the blockage. If infection is the cause, antibiotic drops may be needed.

If the optometrist’s management of the condition is not successful, the patient should be referred to the ophthalmologist, who may carry out further tests including special X-rays. If surgery is needed, a new passage can be created between the tear sac (at the inner corner of the eye) and the inside of the nose; this can be done either using conventional surgical instruments or with the aid of a laser.
Ocular hypertension (OHT)

### Aetiology
Ocular hypertension (OHT) is generally defined as consistently elevated intraocular pressure (IOP) (greater than 21mmHg [2 standard deviations above the population mean IOP] by Goldmann applanation tonometry [GAT] on 2 or more occasions) in one or both eyes in the absence of clinical evidence of optic nerve damage, visual field defect or other pathology that could explain high IOP.

**Aetiology unknown**

The population prevalence estimates for OHT range from 4.5% to 9.4% for those aged > 40 years, with prevalence increasing with age. Data from longitudinal studies indicates that 10% of persons with untreated OHT develop primary open angle glaucoma (POAG) in 5 years.

### Predisposing factors
Increasing age

### Symptoms
Usually asymptomatic

### Signs
- An untreated IOP >21mmHg (GAT), in one or both eyes confirmed on a separate occasion
- Open drainage angles on gonioscopy with normal appearance
- Absence of signs of glaucomatous optic neuropathy (disc changes, field defects)
- Absence of secondary cause for IOP elevation

### Differential diagnosis
- Primary Open Angle Glaucoma (POAG)
- Primary Angle Closure (PAC) or Primary Angle Closure Glaucoma (PACG) (i.e. without or with glaucomatous disc damage)
- Secondary Open Angle glaucoma (e.g. steroid responder, pigment dispersion, pseudo-exfoliation)
- See relevant Clinical Management Guidelines

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

**Non pharmacological**

Guidance on OHT is included in National Institute Health and Care Excellence (NICE) guideline (NG81, November 2017): Glaucoma: diagnosis and management; Scottish Intercollegiate Guidelines Network guideline (SIGN 144, March 2005): ‘Glaucoma referral and safe discharge’ (see Evidence Base); and guidance produced by the College of Optometrists: ‘Examining patients at risk from glaucoma’. NICE guideline NG81 also provides detailed guidance on the diagnosis and management of OHT (including monitoring intervals).

The following guidance on management of OHT is based on NICE NG81, however for optometrists in Scotland SIGN 144 provides specific guidance for referral and management of OHT.

In the case of onward referral for OHT, refer only if IOP is 24mmHg or more using Goldmann-type applanation tonometry. Advise people with IOP below 24 mmHg to continue regular visits to their primary eye care professional (NICE recommendation).

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

For diagnosis of OHT, patients should be offered the following tests: central visual field assessment using standard automated perimetry.
Ocular hypertension (OHT)

Optic nerve assessment and fundus examination using stereoscopic slit lamp biomicroscopy (with pupil dilatation if necessary); optical coherence tomography (OCT) or optic nerve head imaging if available; intraocular pressure (IOP) measurement using Goldmann-type applanation tonometry; peripheral anterior chamber configuration and depth assessments using gonioscopy or, if not available or the patient prefers, the van Herick test or OCT (NICE recommendation) (GRADE*: Level of evidence=low, Strength of recommendation=strong)

People with OHT can be monitored (and treated) by a trained healthcare professional who has all of the following: a specialist qualification in glaucoma; relevant experience; ability to detect a change in clinical status (NICE recommendation). (GRADE*: Level of evidence=low, Strength of recommendation=strong)

At each assessment, re-evaluate risk of conversion to POAG and risk of sight loss to set time to next assessment. (NICE recommendation) (GRADE*: Level of evidence=low, Strength of recommendation=strong)

Data from the Ocular Hypertension Treatment Study (OHTS) and the European Glaucoma Prevention Study (EGPS) have been used to produce a risk calculator to predict the five-year risk of developing POAG in adult patients with ocular hypertension. All of the variables included in the prediction model can be routinely collected in clinical practice, i.e. age; IOP; central corneal thickness (CCT); vertical cup-to-disc (C/D) ratio and pattern standard deviation (PSD). The risk calculator is available online http://ohts.wustl.edu/risk/calculator.html

Offer people the opportunity to discuss their diagnosis, referral, prognosis, treatment and discharge, and provide them with relevant information in an accessible format at initial and subsequent visits (NICE recommendation) (GRADE*: Level of evidence=low, Strength of recommendation=strong)

Pharmacological

Offer a generic prostaglandin analogue to people with an IOP of 24mmHg or more if they are at risk of visual impairment within their lifetime, taking into account risk factors such as: level of IOP, CCT, family history, and life expectancy (NICE recommendation) (GRADE*: Level of evidence=high, Strength of recommendation=strong)

Management Category

B1 (modified): no intervention, routine referral to a consultant ophthalmologist or to an optometrist with a specialist qualification in glaucoma OR
B2: alleviation or palliation, no referral

Possible management by Ophthalmologist

Confirmation of diagnosis
Determination of the individual clinical management plan
Reduction of IOP pharmacologically in patients at moderate or high risk of conversion to POAG
Selective laser trabeculoplasty (SLT)

Evidence base
**GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)**

**Sources of evidence**


For recommendations regarding OHT diagnosis and management, refer to:


**LAY SUMMARY**

If a person has a consistently raised eye pressure but no signs of glaucoma, he or she is said to have Ocular Hypertension (OHT). This is not glaucoma, as there is no damage to the optic nerve (the nerve of sight), but untreated OHT nevertheless leads to Primary Open Angle Glaucoma (POAG) in 10% of patients within five years.

Both the National Institute for Health and Care Excellence (NICE) and the Scottish Intercollegiate Guidelines Network (SIGN) have produced detailed advice on the diagnosis and management of this condition. The optometrist can predict the risk of a patient with OHT developing POAG within five years with the aid of a risk calculator that is available online.

An optometrist who diagnoses a case of OHT and who believes that there is a moderate or high risk of progression to POAG may decide to refer the patient to an ophthalmologist or an optometrist with a specialist qualification in glaucoma. The case can then be re-assessed and, if necessary, pressure-lowering medications or laser surgery can be offered.
### Aetiology
Ocular manifestations of rosacea, a chronic relapsing skin disease of unknown aetiology which often requires long-term management

### Predisposing factors
Rosacea is a common disorder (prevalence up to 10%) with a peak incidence between the fourth and sixth decades of life. It affects females twice as often as males, but the disease can often be more severe in males. Reportedly more common in fair-skinned people of Celtic and Northern European origin

Ocular manifestations occur in 58-72% of patients with rosacea, affecting both sexes equally

Ocular rosacea is most often diagnosed when cutaneous signs and symptoms are present, but it may occur prior to skin involvement (in approx. 20% of cases)

There is no correlation between the severity of the ocular disease and the severity of the cutaneous disease

*Note: rosacea was previously called acne rosacea, a misleading term since the condition is unrelated to acne vulgaris*

### Symptoms
**Ocular symptoms**
- discomfort, irritation, itching, foreign body sensation
- ocular dryness
- photophobia
- blurred vision (if cornea involved)

**Cutaneous symptoms**
- frequent facial flushing (exacerbated by trigger factors) progressing to persistent erythema

Psychological problems including embarrassment, loss of confidence and depression

### Signs
**Ocular signs: lids and tear film**
- hyperaemic thickened lids
- telangiectasia of the lid margins
- chronic posterior marginal blepharitis
- recurrent acute lid infections (chalazion, hordeolum)
- tear film deficiency and/or instability

**Ocular signs: cornea (up to 30% of rosacea patients)**
- punctate staining (fluorescein) of lower third of cornea (usually)
- peripheral vascularisation of inferior cornea
- subepithelial infiltrates around corneal vessels
- sterile ulceration
- corneal thinning (may lead to perforation)
- scarring secondary to corneal involvement
- sclerokeratitis

The Global ROSacea COnsensus Panel (ROSCO) defined a minimum combination of ocular features for a diagnosis of ocular rosacea: lid margin telangiectasia and inter-palpebral injection, or corneal abnormalities, or scleral inflammation

**Cutaneous signs**
- chronic hyperaemia of nose, central forehead and upper cheeks
- telangiectasia of facial blood vessels (permanent distended blood
### Ocular rosacea

- Vessels with a spidery appearance
- Papules, pustules and hypertrophy of sebaceous glands
- Rhinophyma (bullous nose) in severe cases

#### Differential diagnosis
- Tear deficiency
- Interstitial keratitis
- Infectious keratitis
- Other causes of chronic blepharitis

#### Management by Optometrist

**Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.**

<table>
<thead>
<tr>
<th>Non pharmacological</th>
<th>Pharmacological</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Advice on avoiding the causes of exacerbations (including facial flushing) if these have been identified by the patient; can include spicy foods, alcohol, sunlight, heat, cosmetics and soaps (GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
<td>• Ocular lubricants for tear deficiency/instability related symptoms <em>NB: Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Clinical Management Guideline on Conjunctivitis Medicamentosa). They should be switched to unpreserved preparations</em> (GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
<tr>
<td>• Omega 3 fatty acid supplementation (GRADE*: Level of evidence=low, Strength of recommendation=weak)</td>
<td>• Oral antibiotic therapy: doxycycline 40mg modified release once daily for up to 6 weeks (contraindicated in pregnancy and in children under 12 years; various adverse effects have been reported) <em>NB: optometrist prescription of oral antibiotic not recommended unless diagnosis of (cutaneous) rosacea confirmed by dermatologist or GP</em> (GRADE*: Level of evidence=low, Strength of recommendation=weak)</td>
</tr>
<tr>
<td>• Management of associated conditions such as chalazion, hordeolum (stye), posterior marginal blepharitis and tear deficiency or instability (see Clinical Management Guidelines on Blepharitis, Chalazion, Hordeolum, Blepharitis, Dry Eye)</td>
<td></td>
</tr>
</tbody>
</table>

#### Management Category

- **B2**: alleviation/palliation; no referral, but consider co-management with dermatologist or GP
- **A3**: urgent referral to an ophthalmologist if keratitis is severe

#### Possible management by Ophthalmologist

- Topical ciclosporin (unlicensed indication)
- Topical steroid for management of severe corneal disease
- Topical Ivermectin cream to eyelid skin
- Management of corneal perforation: tissue adhesive, lamellar keratoplasty, penetrating keratoplasty
- Restoration of vision lost through corneal disease: penetrating keratoplasty (but high risk of rejection)

### Evidence base

**Ocular rosacea**

Version 9, Page 2 of 4

Date of search 14.02.18; Date of revision 21.08.18; Date of publication 16.10.18; Date for review 13.02.20

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**GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)**

**Sources of evidence**


**LAY SUMMARY**

Rosacea is a common skin disorder, affecting up to one in ten people between the ages of 40 and 60, that can also affect the eye. It causes redness of the nose, forehead and upper cheeks, along with inflammation of the oil glands of the skin. Around a half of rosacea patients have eye involvement, with symptoms of discomfort, dryness and light sensitivity. The optometrist may find inflammation of the eyelids and abnormalities of the tear film (the thin layer of tears covering the surface of the eye) which cause patchy drying of the eye surface. This can cause inflammation of the cornea (the clear window at the front of the eye) with thinning, ulceration, ingrowth of abnormal blood vessels and scarring, all of which can lead to reduced vision.

Dietary advice may help, as may attention to the inflammation of the eyelids. Artificial tears and lubricating ointments may relieve discomfort. An antibiotic given by mouth, usually a drug from the tetracycline family, can improve the condition of both the skin and the eyes. If the condition does not improve, the optometrist may need to refer the patient to a dermatologist.
not respond to simple measures such as these, the optometrist will refer the patient to the ophthalmologist, who may consider prescribing other drugs and may possibly recommend surgery.
Aetiology

Ophthalmia Neonatorum (ON) (conjunctivitis of the newborn) occurs within the first month of life. It is a bacterial, chlamydial or viral infection acquired during passage through an infected birth canal. Since April 2010 it is no longer a notifiable disease in the UK.

Historically, the commonest agent was Neisseria gonorrhoeae (also known as ‘gonococcus’, and a cause of sexually-transmitted disease). The use of silver nitrate drops as prophylaxis was introduced in the C19, although abandoned in the UK in the 1950s. Gonococcal ON develops in approx. 30-50% of newborns exposed to gonococcal infection during delivery.

Nowadays a more usual agent, also sexually acquired by the mother, is Chlamydia trachomatis. Babies born to women with untreated chlamydial infection at delivery have a 30-50% chance of developing ON.

The incubation period is usually as follows:
- C. trachomatis: 5-14 days
- N. gonorrhoeae: 3-5 days

The prevalence of ON differs in different parts of the world and is dependent mainly upon socio-economic conditions, level of knowledge about general health, standard of maternal healthcare as well as the type of prophylactic programme used. UK incidence is:
- C. trachomatis: 6.9 per 100,000 live births
- N. gonorrhoeae: 3.7 per 100,000 live births

In low income countries, very much higher incidences have been reported.

Other bacteria that cause ON include Haemophilus, Streptococcus, Staphylococcus and Pseudomonas species.

Viral infections (less common) can be caused by herpes simplex virus, adenovirus or enterovirus.

The neonatal conjunctiva is particularly vulnerable to infection because of the lack of immunity and the absence of local lymphoid tissue at birth.

Predisposing factors

Infection of the maternal birth canal as the result of sexually-transmitted disease.

This infection may be asymptomatic, especially in the case of C. trachomatis.

Symptoms

(Usually described by mother):
- redness
- discharge (may be profuse in gonococcal infection)
- swelling of lids (may be severe)
- symptoms usually bilateral

Signs

Lids
- oedema (may impede examination of ocular surfaces)

Conjunctival features
### Clinical Management Guidelines

**Ophthalmia neonatorum**

- **mucopurulent conjunctivitis** – discharge may be profuse in *C. trachomatis* infection. *Danger of infection of clinician when prising open lids*
- NB: in neonatal *C. trachomatis* infection there are no follicles as in adults, because of the neonate’s lack of lymphoid tissue
- conjunctival oedema (‘chemosis’)
- conjunctival membrane in severe cases

**Corneal features**
- cornea can be involved, especially in *N. gonorrhoeae* infection. This organism can pass through intact corneal epithelium. Perforation may result
- signs usually bilateral; may be asymmetrical

### Differential diagnosis

- By definition, conjunctivitis occurring within the first month of life is ON
- Congenital obstruction of the nasolacrimal duct(s) is often associated with epiphora, discharge and recurrent conjunctivitis (see Clinical Management Guideline on Nasolacrimal Duct Obstruction)

### Management by Optometrist

**Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere**

<table>
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</table>

**Management Category**

**A1:** emergency (same day) referral to ophthalmologist; no intervention

ON may result in a severe and progressive conjunctivitis with corneal complications and be associated with potentially serious systemic infection

### Possible management by Ophthalmologist

**Diagnosis**

- conjunctival cultures for bacteria (*N. gonorrhoeae* requires special media)
- conjunctival scraping for Gram stain (bacteria) and Giemsa stain (for *C. trachomatis*)
- Polymerase Chain Reaction (PCR) studies

**Treatment**

**Bacterial conjunctivitis**

- systemic penicillin G or a cephalosporin for *N. gonorrhoeae*
- topical erythromycin sometimes given in addition
- other topical antibiotics, including azithromycin or chloramphenicol
- frequent irrigation until discharge ceases

**Chlamydial conjunctivitis**

- systemic erythromycin
- topical azithromycin

**Herpetic conjunctivitis**

- systemic and topical antiviral, e.g. aciclovir

### Evidence base
LAY SUMMARY

The definition of Ophthalmia Neonatorum (conjunctivitis of the newborn) is an eye infection that occurs within the first 30 days of life. It is caught during birth by contact with the mother’s birth canal that is infected with a sexually-transmitted disease. The infection may be bacterial, chlamydial or viral. Historically, gonorrhoea was the usual cause, but chlamydial infection is now more common. Globally, the prevalence of this infection varies widely according to prevailing socio-economic conditions, health education and maternal healthcare.

The baby’s eyes are red, the eyelids and the whites of the eyes are swollen and there is watering or a discharge. Usually both eyes are affected, but one may be worse than the other. One of the dangers of gonorrhoeal infection is that it may affect the cornea also. Early diagnosis is important and for this reason, the optometrist is advised to refer all cases immediately to the on-call ophthalmologist. Swabs will be taken for culture and treatment started without delay, using antibiotics given by mouth or by injection or into a vein, and in eye drop form.

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence


### Aetiology

Infestation of lid margins by the crab louse (*Phthirus pubis*), a blood-feeding obligate ectoparasite affecting only humans

- (NB in Pediculosis capitis, i.e. infestation by head lice [*Pediculus humanus capitis*], the lashes are rarely involved)
- crab lice infest coarsely spaced hair, predominantly pubic hair
- lashes also provide ideal spacing
- genital-to-eye transmission, possibly on bedding and towels
- crab lice survive no more than two days if separated from human host
- this insect is not a vector for other diseases
- in severe cases, lice faeces can cause keratoconjunctivitis

### Predisposing factors

Most common in sexually active people between 15 and 45 years old

Can be contracted within families through poor hygiene

### Symptoms

Intense itching of lid margins

Red watery eye

Unilateral or bilateral

### Signs

- madarosis (loss of lashes)
- conjunctival hyperaemia
- superficial punctate keratopathy (SPK)
- bites leave red inflamed areas on lid margins
- possible pre-auricular lymphadenopathy
- adult lice (1.0–1.5mm long) attached to lash; almost completely transparent (high magnification [x40] required at slit lamp)
- eggs (termed nits) in greyish white cigar-shaped shells attached near base of lashes. Empty shells remain after hatching
- reddish-brown deposits at the base of the lashes are a mixture of louse faeces and host blood following louse bites

### Differential diagnosis

Blepharitis (anterior)

- nits may be confused with lash debris
- *Demodex* mites are much smaller than crab lice (0.1–0.4mm long) and are not usually seen outside the lash follicle

Allergic reactions affecting lid skin

### Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

**Non pharmacological**

Sensitive counselling (i.e. by GP) required as this is a sexually transmitted disease

- advice on personal hygiene: wash hands after touching pubic region
- NB possibility of sexual abuse of children

Remove lice, nits and shells (casts) at slit lamp

- use forceps (lice have a tenacious grip on the lashes)

Advise on any symptoms of pubic infestation

- effective treatments (e.g. malathion, permethrin) available without prescription from pharmacies

Sexual partners or family members at risk should have their eyes examined and treated if necessary
Bed linen, towels and clothes should be washed at 60°C for at least 5 min  
(GRADE*: Level of evidence=low, Strength of recommendation=strong)

**Pharmacological**

Application of Simple Eye Ointment to the lid margins will suffocate lice  
(unmedicated ointment, applied twice daily for at least 2 weeks)  
(GRADE*: Level of evidence=low, Strength of recommendation=strong)

Permethrin 1% lotion applied to lashes for 10 minutes with eyes closed  
NB: insecticides can be toxic to the cornea  
(GRADE*: Level of evidence=low, Strength of recommendation=weak)

**Management Category**

Referral via GP for management of non-ocular aspects, including tracing and screening close contacts; also screening for other sexually-transmitted diseases  
**B1** (modified): possible prescription of drugs: telephone GP to discuss referral and for advice on local safeguarding arrangements in the case of a child

**Possible management by Ophthalmologist**

Heavy infestation can be reduced by Argon laser photo-ablation or cryotherapy (freezing)

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence


**LAY SUMMARY**

This condition is caused by infestation of the eyelashes by the crab louse, which is usually acquired by sexual contact.

Phthiriasis causes the eyelids to become itchy and the eyes to become red and watery. It may be possible to see the eggs of the lice (called nits) clinging to the bases of the eyelashes.
The treatment involves removing the lice and nits at the slit lamp (the clinical microscope used by optometrists and ophthalmologists). Pubic infection can be treated with drugs available without prescription from pharmacies. Patients are advised that they have a sexually transmitted condition and that their partner(s) may need to be examined also. Bed linen, towels and clothes should be washed at 60°C for at least five minutes.
### Aetiology
Degenerative conjunctival lesion, usually situated nasally at the limbus
Degeneration of collagen fibres of the conjunctival stroma
- hyalinisation and granular deposits
- thinning of overlying epithelium
- occasional calcification

### Predisposing factors
Increasing age (seen in most eyes by age 70)
Published figures of prevalence range from 11-75% (prevalence depends on age and geographical location of the sample)
Long term exposure to UV radiation
- sunlight (residence at or near the equator, outdoor work, especially on reflective surfaces e.g. sand, concrete, water, snow)
- welding and other occupational exposure
Male gender (likely to be related to occupational exposure)
Chronic irritation from wind or dust
Contact lens wear

### Symptoms
Usually asymptomatic
Possible mild foreign body sensation and redness when inflamed
Occasional cosmetic concern

### Signs
Area of conjunctival thickening adjoining the limbus
- in the palpebral aperture, usually at 3 & 9 o’clock positions
- more common nasally
- usually bilateral
Elevated and less transparent than normal conjunctiva
White to yellow colour, fat like appearance, calcification sometimes present
Sometimes slightly more hyperaemic than surrounding conjunctiva
May become inflamed (pingueculitis) causing mild ocular irritation
May lead to Dellen in adjacent cornea
Decreased TBUT

### Differential diagnosis
Pterygium
- easily distinguished because pinguecula does not cross the limbus to involve the cornea
- pinguecula does not progress to become pterygium; they are two distinct conditions
Conjunctival intraepithelial neoplasia (can resemble a keratinised pinguecula)
Dermoid cyst
Epithelial retention cyst (thin-walled lesion containing clear fluid)
Differentiate from inflammatory conditions, e.g. episcleritis, angular conjunctivitis

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

| Non pharmacological | Reassure patient about benign nature of the lesion (no threat to health or sight)
| Advise on UV protection to minimise risk of inflammation
| Cold compresses when inflamed
| (GRADE*: Level of evidence=low, Strength of recommendation=strong) |

| Pharmacological | Ocular lubricants for symptomatic relief (drops for use during the day, unmedicated ointment for use at bedtime |
NB Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Guideline on Conjunctivitis Medicamentosa). They should be switched to unpreserved preparations

GRADE*: Level of evidence=low, Strength of recommendation=strong

Pingueculitis usually responds to a brief course of a 'non-penetrating’ topical steroid (e.g. fluorometholone, loteprednol) or a topical non-steroidal anti-inflammatory drug (off-licence use)

NB All patients on topical steroid drops or ointment should have their intraocular pressures checked initially, then measured again at 2 weeks and every 4 weeks for 2-3 months (see Clinical Management Guideline on Steroid Glaucoma)

GRADE*: Level of evidence=moderate, Strength of recommendation=weak

Management Category B2: Alleviation / palliation: normally no referral

Possible management by Ophthalmologist

Excision is very rarely warranted

A single case series has described effective cosmetic removed of pingueculae by argon laser photocoagulation

Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence


LAY SUMMARY
A pinguecula is a small raised spot, white to yellowish in colour, that sometimes appears on the surface of the eye at the limbus. The limbus is where the white of the eye (the sclera) and the transparent window at the front of the eye (the cornea) meet. If the cornea is imagined as a clock
face, a pinguecula will generally form at the three and nine o'clock positions. This condition becomes more common as people age, so that by 70 years most people have them. Both eyes are usually affected. There is no effect on vision.

This is a mild degenerative condition, due to long-term exposure to ultra-violet (UV) light, either occurring naturally in sunlight or artificially in some occupations. A pinguecula usually causes no symptoms, but if it becomes inflamed it may cause local redness of the eye and irritation or discomfort. Sometimes people complain of the cosmetic appearance.

The optometrist will examine the pinguecula carefully, distinguishing it from other small spots and cysts that sometimes appear on the eye surface in this position. Once the diagnosis is made, the patient will be advised to limit UV exposure by wearing a hat and sunglasses when it is sunny. If the pinguecula becomes inflamed, anti-inflammatory eye drops are sometimes recommended. Sometimes patients ask for a pinguecula to be removed, which can be done by surgery or laser treatment. As this is nearly always a cosmetic procedure, it is rarely undertaken.
## Post-operative suture breakage

**Aetiology**
Breakage of a suture or sutures remaining after surgery, usually with protrusion of broken end or knot

| Predisposing factors | Corneal transplant sutures (usually 10/0 monofilament nylon)  
• continuous suture: single suture sewn in zigzag pattern alternately between the transplant and host. Usually left *in situ* for a year or more  
• interrupted sutures: usually 16 (range 8-24) sutures. Individual interrupted sutures, or diametrically opposite pairs of sutures, are sometimes removed earlier for control of astigmatism. Most usually left *in situ* for a year or more  
Sutures used in other surgery (usually either nylon or vicryl)  
• cataract, glaucoma, pterygium, squint, vitreoretinal procedures  
Even if their material is inert, all sutures can cause irritation when loose or broken and this can predispose to inflammation and infection |

### Symptoms
One or more of the following may be present:

- foreign body sensation
- irritation
- redness
- photophobia
- epiphora
- alteration in visual acuity following changes in astigmatism

### Signs
One or more of the following may be present:

- suture end may be visible
- discharge – may be purulent, if infected
- injection ± corneal vascularisation (enhanced risk of transplant rejection)
- corneal infiltrate around suture ± corneal abscess ± hypopyon
- uveitis: flare, cells and KPs. Intraocular pressure may be raised
- mucus filaments
- conjunctival hyperaemia
- papillae on overlying tarsal conjunctiva (more common in chronic irritation)
- wound leak (Seidel positive if fluorescein pool [2% Minims] appears diluted as aqueous oozes from site of leak [use cobalt blue filter])

**Differential diagnosis**  
Foreign body (corneal or sub-tarsal)  
Allergic conjunctivitis  
Idiopathic acute anterior uveitis

### Management by Optometrist
 Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

#### Non pharmacological
Caution should be exercised if it is planned to remove a loose or broken corneal transplant or trabeculectomy suture without prior discussion with the HES  
Sterile instruments must be used  
The wound should be checked for leakage after suture removal, using the Seidel test  
(GRADE*: Level of evidence=low, Strength of recommendation=weak)

#### Pharmacological
Topical anaesthetic (gutt. proxymetacaine 0.5% or gutt. oxybuprocaine 0.4%) may be necessary to aid examination and for suture removal  
Topical lubricant (gel-based or ointment) for symptomatic relief
(GRADE*: Level of evidence=low, Strength of recommendation=strong)

If there is a likelihood of infection, consider topical antibiotic prophylaxis (e.g. gutt. chloramphenicol 0.5% qds for 5 days)

(GRADE*: Level of evidence=low, Strength of recommendation=weak)

**Management Category**

<table>
<thead>
<tr>
<th>A3: urgent (within one week) referral to ophthalmologist</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1: emergency (same day) referral to ophthalmologist if:</td>
</tr>
<tr>
<td>• evidence of infection / inflammation</td>
</tr>
<tr>
<td>• wound leak present</td>
</tr>
<tr>
<td>• broken corneal transplant suture (risk of rejection)</td>
</tr>
</tbody>
</table>

| B2 (modified): suture removal, preferably following discussion with the HES |

**Possible management by Ophthalmologist**

- Removal of offending suture, followed by topical antibiotic ± steroid
- Possibly send for microbiology/histology
- Anterior chamber tap/vitreous tap if required
- Re-suturing if necessary

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://www.gradeworkinggroup.org/index.htm)

Sources of evidence

None applicable

**LAY SUMMARY**

It sometimes happens that, following surgery to the eye, a suture (stitch) breaks. If this happens, the eye can become uncomfortable and it may feel as if there is a piece of grit in the eye. The eye may also water excessively, become red, unduly sensitive to light and sometimes the vision is affected. Depending on the severity of the case, the optometrist will usually refer the patient either urgently or as an emergency to the ophthalmologist, who will trim or remove the broken suture(s).
### Aetiology
Fibrovascular growth progressing from the bulbar conjunctiva to involve the cornea
Possibly a tissue response to irritants rather than a true degeneration
The prevalence of pterygium varies from 1.2% in Caucasians of urban, temperate climates to 23.4% in the black population of tropical Barbados
Multifactorial aetiology, most popularly attributed to chronic exposure to UV light, dust and wind. The human papillomavirus has also been implicated

### Predisposing factors
- Older age
- Male gender (probably related to occupational exposure)
- Long term exposure to ultraviolet radiation
  - sunlight (residence at or near the equator, outdoor work, especially on reflective surfaces e.g. sand, concrete, water, snow)
- Dryness, arid climate

### Symptoms
- Mild irritation (redness, dryness, foreign body sensation)
- May be exacerbated by incidents of acute inflammation
- Cosmetic concern

### Signs
- Usually bilateral; often asymmetrical. More common nasally
- Starts with scarring, thickening and distortion of the bulbar conjunctiva
- Small grey corneal opacities appear near the limbus
- Conjunctiva overgrows these opacities
- Slow insidious growth on to cornea (or may become stable)
- Destroys Bowman’s membrane and superficial stroma lamellae
- Epithelial iron deposit (Stocker’s line) ahead of advancing pterygium
- Relatively rich surface vascularisation
- Flattening of cornea in horizontal meridian

### Differential diagnosis
- Pingeula (no corneal involvement)
- Pannus
- Pseudopterygium
  - adhesion of a fold of conjunctiva to a peripheral corneal ulcer, which is fixed only at its apex to the cornea (pterygium is adherent to underlying structures throughout)
- Carcinoma in situ of the cornea or conjunctiva (also known as Bowen’s disease and as intraepithelial squamous cell carcinoma)

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

#### Non pharmacological
Advising on UV protection:
- brimmed hat, tinted lenses, wrap-around style for side protection
- reduces risk of progression and of becoming inflamed and irritated
  (GRADE*: Level of evidence=low, Strength of recommendation=strong)

Measure and draw diagram (photodocument if possible)
Cold compress when inflamed
  (GRADE*: Level of evidence=low, Strength of recommendation=strong)

#### Pharmacological
Ocular lubricants for symptomatic relief (drops for use during the day, unmedicated ointment for use at bedtime)
*NB Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Clinical Management Guideline on Conjunctivitis Medicamentosa). They should be switched to*
Acute inflammation of a pterygium usually responds to a brief course of a 'non-penetrating' topical steroid (e.g. fluorometholone, loteprednol) or a topical non-steroidal anti-inflammatory drug (off-licence use)

NB All patients on topical steroid drops or ointment should have their intraocular pressures checked initially, during and at the end of treatment (see Clinical Management Guideline on Steroid Glaucoma)

GRADE*: Level of evidence=moderate, Strength of recommendation=weak

Management Category

B2: alleviation/palliation; normally no referral
B1: refer to ophthalmologist if pterygium:
  • threatens visual axis
  • induces irregular astigmatism
  • is associated with chronic inflammation
  • is cosmetically unacceptable

Possible management by Ophthalmologist

Acute inflammation
  • topical steroids
  • non steroidal anti-inflammatory agents

A variety of surgical techniques, including:
  • surgical excision (bare sclera resection)
  • surgical excision plus conjunctival closure, conjunctival flap closure or conjunctival autografting; or amniotic membrane grafting
  • adjunctive treatment sometimes given; includes:
    – beta irradiation (post-op)
    – topical thiopeta (post-op)
    – mitomycin C (intra- or post-op)

A recent systematic literature review concluded that bare sclera resection was associated with high rates of recurrence. By contrast, conjunctival or limbal autograft was associated with lower rates of recurrence

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

Sources of evidence

Clearfield E, Muthappan V, Wang X, Kuo IC. Conjunctival autograft for pterygium. Cochrane Database of Systematic Reviews 2016;2:CD011349


LAY SUMMARY
A pterygium is a triangular thickening of the conjunctiva (the layer of transparent skin on the white of the eye) which extends on to the outer edge of the cornea (the transparent window at the front of the eye). If the cornea is imagined as a clock face, a pterygium normally occurs at the three and nine o’clock positions, more usually on the nasal side. A pterygium may grow over the corneal surface. Because of tissue shrinkage, it can put tension on the cornea, causing astigmatism (loss of spherical curvature) and reducing the sharpness of vision. Patients may complain of irritation of the affected eye and they may be concerned about the cosmetic appearance.

Pterygium is caused by long-standing exposure of the eyes to ultra-violet (UV) light, dust and wind. Because of this, it is commoner near the equator, where UV levels are higher, than in temperate parts of the world.

Having carefully examined the affected eye(s) the optometrist will record the dimensions of the pterygium for future comparison. Artificial tears and lubricating ointment may be enough to control the irritation of the eyes. If the pterygium is inflamed, a short course of steroid eye drops may be prescribed. If the pterygium continues to grow towards the middle of the cornea, threatening the vision of the eye, or if inflammation cannot be controlled, the patient will be referred to the ophthalmologist. Various different surgical techniques are available if the pterygium needs to be removed.
Recurrent corneal epithelial erosion syndrome

### Aetiology
- Recurrent breakdown of corneal epithelium due to defective adhesion to basement membrane
- Prevalence greatest between third and fourth decade
- Initial cause may have been traumatic, but underlying epithelial dystrophy may be present
- Repair of epithelial basement membrane takes around three months if largely undisturbed

### Predisposing factors
- History of superficial trauma
- Corneal dystrophy (especially Map-Dot-Fingerprint Dystrophy [Epithelial Basement Membrane or Cogan’s Dystrophy])
- Posterior marginal blepharitis (Meibomian gland dysfunction)
- Diabetes
- Previous refractive surgery (particularly PRK)

### Symptoms
- Unilateral sharp pain, typically sudden onset on waking and opening eyes; may also awake patient in middle of night
- Feeling as if eyelid is stuck to eyeball
- Lacrimation
- Photophobia
- Blurred vision
- May recur over weeks, months or years

### Signs
- Epithelial erosion (usually inferior cornea)
  - stains with fluorescein
  - ‘loose’ edges, ‘slipped rug’ appearance
- Intra-epithelial microcysts
- Mild stromal oedema
- NB: examine both eyes for signs of corneal dystrophy

### Differential diagnosis
- Tear deficiency
- Other corneal dystrophies with epithelial manifestations
- Contact lens-related epithelial conditions

### Management by Optometrist

#### Non pharmacological
- Bandage contact lens (although trials suggest that bandage lenses are equivalent to lubrication alone)
  (GRADE*: Level of evidence=moderate, Strength of recommendation=weak)

#### Pharmacological
- Mild cases:
  - ocular lubricants
    (GRADE*: Level of evidence=low, Strength of recommendation=strong)
  - artificial tears (preferably unpreserved) frequently during day
  - unmedicated ointment (e.g. oc Lacrilube) before sleep – should be continued for at least 3 months from date of last recurrence (however, one study showed that the use of unmedicated ointment at night for two months following traumatic corneal abrasions led to increased symptoms of recurrent corneal erosion)
  (GRADE*: Level of evidence=low, Strength of recommendation=weak)
  - review at monthly intervals for three months. Advise patient to return/seek further help if symptoms persist
- More severe cases with large area of epithelial loss:
  - cycloplegic agent (e.g. gutt. cyclopentolate 1%) to prevent pupil
Recurrent corneal epithelial erosion syndrome

**Management Category**

<table>
<thead>
<tr>
<th>Management Category</th>
<th>Possible management by Ophthalmologist</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>B2:</strong> alleviation/palliation, normally no referral</td>
<td>For those not responding to medical therapy:</td>
</tr>
<tr>
<td></td>
<td>• débridement of loose epithelium</td>
</tr>
<tr>
<td></td>
<td>• excimer laser photo-therapeutic keratectomy</td>
</tr>
<tr>
<td></td>
<td>• micropuncture with hypodermic needle or YAG laser</td>
</tr>
<tr>
<td></td>
<td>• 'alcohol delamination'</td>
</tr>
<tr>
<td></td>
<td>• diamond burr polishing of Bowman’s membrane</td>
</tr>
</tbody>
</table>

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*

**Sources of evidence**


**LAY SUMMARY**

In this condition the surface skin of the cornea (the clear window of the eye) breaks down, causing sharp pain, watering and sometimes blurred vision. This may happen as the patient wakes after sleep. It may be due to a previous mild injury (corneal abrasion) or to a condition known as a dystrophy in which the surface of the cornea is unusually delicate. The condition may recur over weeks or months. It is treated by reducing friction between the eye and the eyelids, using lubricating drops and/or ointments, to encourage complete healing of the eye surface. Sometimes
other measures are needed, for example a special contact lens applied as a bandage, minor surgery or laser therapy.
Scleritis

Aetiology

Scleritis is a potentially severe inflammatory disease of the sclera which is bilateral in 50% of cases.

Predisposing factors

Patients are usually in the middle age group (40-60 years) M:F = 2:3
May be idiopathic, but approximately a third of cases are associated with systemic inflammatory disease, of which the scleritis may be the first presentation:
- rheumatoid arthritis
- granulomatosis with polyangiitis (formerly known as Wegener’s granulomatosis)
- systemic lupus erythematosus
- polyarteritis nodosa
- inflammatory bowel disease
- syphilis
- sarcoidosis, TB

Local causes: e.g. herpes zoster, trauma, surgery (surgery-induced necrotizing scleritis, SINS)

Symptoms

Moderate or severe pain (eye ‘ache’ may be referred to brow or jaw) which is exacerbated by eye movement
May disturb sleep
Gradual onset
Tenderness of globe
Photophobia
Epiphora
Visual loss
Possible history of previous episodes

Signs

Scleritis may involve the anterior sclera, the posterior sclera, or both

**Anterior scleritis** (90% of cases)
(a) Non-necrotising (75% of cases)
- usually unilateral
- hyperaemia of superficial and deep episcleral vessels; does not blanch with vasoconstrictors (e.g. gutt. phenylephrine 2.5%)
- anterior uveitis may be present
- tenderness of globe
- when inflammation resolved, choroidal pigment may show through thinned sclera as a blue/black colouration
- approximately 60% are diffuse and 40% nodular (scleral nodule cannot be moved over underlying tissue)
(b) Necrotising (15% of cases)
- the most severe form (may occur in the absence of pain). 75% will eventually have visual impairment
- avascular patches leading to scleral melting with ectasia and choroidal herniation

**Posterior scleritis** (10% of cases)
Involves sclera posterior to the ora serrata. Eye may be white.
Ophthalmoscopy may show exudative retinal detachment, macular oedema, optic disc oedema, but may also show no abnormality

### Differential diagnosis
- Episcleritis (see Clinical Management Guideline on Episcleritis)
- Other causes of acute red eye

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

<table>
<thead>
<tr>
<th>Non pharmacological</th>
<th>None</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharmacological</td>
<td>Systemic analgesia (e.g. paracetamol, aspirin, ibuprofen)</td>
</tr>
<tr>
<td></td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

### Management Category
- **A2**: First aid measures and emergency (same day) referral. Telephone on-call ophthalmologist. No intervention except analgesia. Scleritis is potentially sight-threatening

### Possible management by Ophthalmologist
- Investigation for systemic associations
- Systemic non-steroidal anti-inflammatory drugs
- Topical steroid
- Systemic immunosuppression (corticosteroids +/- other immunosuppressant drugs, and biologics, e.g. TNFalpha inhibitors)
- Imaging to investigate posterior segment involvement
- May require referral to, or co-management with, Physician or Rheumatologist

### Evidence base
*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)

**Sources of evidence**


LAY SUMMARY

Scleritis is a rare, severe inflammation of the sclera (the white part of the eyeball). It affects older people, women more than men, around a third of whom have some other form of inflammation, such as rheumatoid arthritis or inflammation of the bowel, or long-standing infection elsewhere in the body. It may affect one or both eyes.

The condition begins gradually and patients experience an aching pain in the eye, which may spread to the brow region or to the jaw. This may be so severe as to cause loss of sleep. Patients may also find lights unbearably bright and the vision may be blurred.

There are two forms of Scleritis: Anterior Scleritis, which makes up 9 out of 10 cases and involves the sclera of the front part of the eye, where the inflammation (redness and swelling) can easily be seen. An even more severe form of Anterior Scleritis seen in 15% of these cases is known as Necrotising Scleritis, in which the scleral tissue melts away, often without pain; vision is likely to be greatly impaired. One in 10 cases of Scleritis takes the form known as Posterior Scleritis, which affects the sclera of the back part of the eye, so that the front of the eye may appear normal and the optometrist will need to use special instruments to help to make the diagnosis.

Scleritis is a serious condition and it is recommended that all cases be referred as emergencies to the ophthalmologist, who will usually treat the condition with drugs given by mouth that reduce inflammation and suppress the body’s immune system.
Sub-conjunctival haemorrhage

**Aetiology**
Spontaneous in 50-87% of cases; may be recurrent
Known causes include:
- Valsalva manoeuvre (e.g. coughing, straining, vomiting) producing rise in central venous pressure
- Traumatic (may be isolated or associated with ruptured globe or retrobulbar haemorrhage – see Clinical Management Guideline on Blunt Trauma)
- Recent eye surgery

**History is important.** Ask about hypertension, medications, acute or chronic cough, eye rubbing, heavy lifting, recent ocular or head trauma, bleeding or clotting abnormalities and recurrent subconjunctival haemorrhage

**Predisposing factors**
- Older age (highest incidence at 60-80 years)
- Trauma (including contact lens-related injury)
- Systemic hypertension
- Anticoagulant medication (e.g. aspirin, warfarin)
- Diabetes and other systemic vascular disorders
- Bleeding abnormality (leukaemia, clotting disorders)
- Long-term topical steroid treatment
- Conjunctival vascular lesion

**Symptoms**
- Mild ache or irritation (no pain)
- May be asymptomatic

**Signs**
- Red area on eye, location usually inferior, caused by blood beneath the conjunctiva of which the posterior border can be seen (if cannot be seen, may originate from intra-cranial haemorrhage, in which case immediate emergency referral may save a life)
- Usually unilateral
- No discharge

**Differential diagnosis**
- Haemorrhagic conjunctivitis (EHC)
  - Viral conjunctivitis (usually small multiple haemorrhages; rare)
  - Usually bilateral
- Conjunctival neoplasms (e.g. lymphoma) with secondary haemorrhage
- Kaposi’s sarcoma (red or purple lesions under conjunctiva)

**Management by Optometrist**
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

**Non pharmacological**
- Measure blood pressure (see NICE guidance at: [http://www.nice.org.uk/guidance/cg127/chapter/1-guidance - measuring-blood-pressure](http://www.nice.org.uk/guidance/cg127/chapter/1-guidance - measuring-blood-pressure))
- In traumatic cases, refer to Clinical Management Guideline on Blunt Trauma
- Ensure that posterior border of haemorrhage can be seen, to exclude intra-cranial source e.g. following skull base fracture
  (GRADE*: Level of evidence=low, Strength of recommendation=strong)

  If patient has history of recurrent subconjunctival haemorrhages or a history of bleeding or clotting abnormalities, refer to GP. Also refer for checking of international normalized ratio (INR) if patient is on warfarin
**CLINICAL MANAGEMENT GUIDELINES**

**Sub-conjunctival haemorrhage**

(partially if associated with unexplained bruising on the skin)
- reassure patient
- condition usually clears within 5-10 days
- cold compress may reduce discomfort

Advise patient to return/seek further help if problem does not resolve or if it recurs

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

<table>
<thead>
<tr>
<th>Pharmacological</th>
<th>Tear supplement / ocular lubricant if mild ocular irritation is present</th>
</tr>
</thead>
<tbody>
<tr>
<td>Management Category</td>
<td>B3: management to resolution</td>
</tr>
<tr>
<td>Refer to GP if suspicion of hypertension or bleeding disorder, or if condition is recurrent</td>
<td></td>
</tr>
<tr>
<td>A1: if intracranial source of haemorrhage suspected, emergency (same day) referral to A&amp;E</td>
<td></td>
</tr>
</tbody>
</table>

**Possible management by Ophthalmologist**

(Not normally referred)
- Investigate for underlying cause of subconjunctival haemorrhage
- Cauterise bleeding vessel if found

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see [http://gradeworkinggroup.org/toolbox/index.htm](http://gradeworkinggroup.org/toolbox/index.htm))

**Sources of evidence**


**LAY SUMMARY**

Sub-conjunctival haemorrhage

Version 11, Page 2 of 3

Date of search 26.02.19; Date of revision 24.04.19; Date of publication ab.cd.ef; Date for review 25.02.21

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Sub-conjunctival haemorrhage

Sub-conjunctival haemorrhage (S-CH) is a common condition which is usually unimportant but very occasionally indicates a serious medical condition. It occurs when a small amount of bleeding takes place beneath the conjunctiva (the membrane overlying the white of the eye) and is similar to a bruise elsewhere. It appears bright red because the conjunctiva is transparent. This may happen spontaneously (that is, with no apparent cause) or as the result of minor injury, for example when a contact lens is mishandled. It can also indicate raised blood pressure or a bleeding abnormality. S-CH occurs more often in people taking blood thinning medications or aspirin, and in diabetics. The condition is often alarming because of its dramatic appearance but there is usually only mild discomfort and the haemorrhage usually disappears in 5-10 days without treatment. It is usual to check the blood pressure of people with S-CH and to investigate the problem if it recurs.
Dry Eye Disease (Keratoconjunctivitis Sicca, KCS)

### Aetiology

The 2017 International Dry Eye Workshop (DEWS II) has provided the following definition: Dry eye is a multifactorial disease of the ocular surface characterised by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play aetiological roles.

Aetiology: Dry Eye Disease (DED) is divided into aqueous-deficient dry eye and evaporative dry eye, and the two forms can occur together. The lists that follow are simplified from TFOS DEWS II:

1. Aqueous-deficient Dry Eye (ADDE)
   - Sjögren Syndrome Dry Eye (SSDE)
     - associated systemic diseases, e.g. rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, granulomatosi with polyangiitis (formerly known as Wegener’s granulomatosis)
   - Non-Sjögren Syndrome Dry Eye (NSDE)
     - intrinsic lacrimal gland deficiency
     - age-related dry eye
     - inflammation or infiltration of lacrimal gland
       - sarcoidosis
       - lymphoma
       - viral infection
       - radiation injury
     - lacrimal gland obstruction
       - cicatricial conjunctivitis
         - Graft Versus Host Disease (GVHD)
         - Stevens-Johnson syndrome (SJS)
         - cicatricial pemphigoid
         - trachoma
         - chemical injury
       - hyposecretory states
         - reflex afferent block
           - topical anaesthesia
           - trigeminal nerve injury
             - refractive surgery
             - neurotrophic keratitis
         - secretomotor block
           - parasympathetic damage
           - pharmacological inhibition (wide range of systemic drugs, including antidepressants, anticholinergics, antipsychotics, antihistamines, chemotherapeutic agents, antihypertensives, anti-arrhythmics, antithyroid agents and opioid analgesics)

2. Evaporative Dry Eye (EDE)

Tear deficiency can also be related to tear film dysfunction:

- Meibomian gland dysfunction
- Secondary to local disease
  - anterior blepharitis
Dry Eye Disease (Keratoconjunctivitis Sicca, KCS)

- ocular surface inflammation

  - Secondary to systemic disease
    - rosacea
    - seborrhoeic dermatitis
    - atopic dermatitis
    - ichthyosis
    - psoriasis

  - Genetically determined Meibomian gland disorders

  - Lid aperture disorders
    - thyroid eye disease
    - ectropion
    - abnormal blink

  - Ocular surface-related EDE
    - allergic eye disease
    - vitamin A deficiency
    - iatrogenic disease including contact lens wear

<table>
<thead>
<tr>
<th>Predisposing factors</th>
<th>Wide variation in prevalence worldwide (6.5% to 52.4%); higher prevalence in women in all studies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Wide variation in prevalence by symptom compared to clinical diagnosis</td>
</tr>
<tr>
<td></td>
<td>Prevalence rises with age, between 2.0% and 10.5% per decade</td>
</tr>
<tr>
<td>Factors that aggravate symptoms</td>
<td>• noxious agents (cooking fumes, tobacco smoke)</td>
</tr>
<tr>
<td></td>
<td>• increased evaporation of tears (air conditioning, central heating)</td>
</tr>
<tr>
<td></td>
<td>• digital device use (reduced blink interval)</td>
</tr>
<tr>
<td></td>
<td>• contact lens wear</td>
</tr>
<tr>
<td></td>
<td>• conjunctivitis medicamentosa secondary to long-term topical therapy</td>
</tr>
</tbody>
</table>

| Symptoms | • ocular irritation                           |
|          | • foreign body, gritty or burning sensation   |
|          | • presence of a stringy mucous discharge      |
|          | • blurring of vision from epithelial disruption or (transiently) from mucus strands          |
|          | • symptoms exacerbated by smoke, wind or heat |
|          | • symptoms usually bilateral; may not be described as a feeling of dryness                  |
|          | • associated symptoms of dry mouth, systemic disease (e.g. arthritis)                        |

| Signs | • reduced tear meniscus at inferior lid margin (following the instillation of fluorescein, normal meniscus is not less than 0.2 mm in height) |
|       | • raised tear osmolarity (308 mOsm/l is the most sensitive threshold to distinguish normal from mild/moderate DED, while 315 mOsm/l is the most specific cut-off) |
|       | • fluorescein break up time (FBUT) <10 sec                                                  |
|       | • Schirmer test (without anaesthesia) ≤ 5 mm in 5 min                                        |
|       | • punctate epithelial erosions in exposed area of cornea and bulbar conjunctiva (especially in inferior third of palpebral aperture). Stain with vital dye(s) as available. Various grading systems are available (e.g. Oxford staining score) |
|       | • lid wiper epitheliopathy                                                                  |
|       | • increased mucus strands and other tear film débris                                        |
|       | • filaments (adherent comma-shaped mucus strands)                                          |
### Differential diagnosis
- Anterior blepharitis
- Allergic and infective conjunctivitis
- Eyelid abnormality or dysfunction leading to exposure (exposure keratoconjunctivitis)
- Nocturnal lagophthalmos (failure to close eyes at night)

### Management by Optometrist

**Non pharmacological**
- Patient education regarding the condition
- Modification of local environment
  - desiccating conditions and environmental pollutants
  - digital device use
  (GRADE*: Level of evidence=low, Strength of recommendation=strong)
- Advise *diet* rich in omega-3 essential fatty acids
  (GRADE*: Level of evidence=low, Strength of recommendation=strong)
- Oral essential fatty acid *supplements* (omega-3 and omega-6): a recent high-quality RCT found that patients who were randomly assigned to receive supplements containing 3000mg of omega-3 fatty acids for 12 months did not have significantly better outcomes than those who were assigned to receive placebo
  (GRADE*: Level of evidence=moderate, Strength of recommendation=weak)
- Tear conservation
  - diminish outflow – punctal plugs
  (GRADE*: Level of evidence=moderate, Strength of recommendation=strong)
- Lid hygiene for meibomian dysfunction (hot compresses, lid hygiene) (see Clinical Management Guideline on Blepharitis)
  (GRADE*: Level of evidence=moderate, Strength of recommendation=strong)
- Protection with therapeutic contact lenses
  (GRADE*: Level of evidence=low, Strength of recommendation=weak)

**Pharmacological**
- Tear supplements (preferably unpreserved) for use during the day ± unmedicated ointment for use at bedtime
  (Recent systematic review found no evidence to support the superiority of any particular tear supplement)
- Liposomal sprays in evaporative dry eye
  (GRADE*: Level of evidence=moderate, Strength of recommendation=strong)

*NB Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Clinical Management Guideline on Conjunctivitis Medicamentosa). They should be switched to unpreserved preparations*

- Topical steroids (such as fluorometholone or loteprednol) may be considered for
Dry Eye Disease (Keratoconjunctivitis Sicca, KCS)

Management Category

<table>
<thead>
<tr>
<th>Management Category</th>
<th>B2: alleviation or palliation; normally no referral (If idiopathic and not associated with systemic disease)</th>
</tr>
</thead>
</table>

**B1:** initial management followed by routine referral if adequate trial of topical treatment or punctal plugs fails, or for secondary complications (vascularisation, corneal scaring, melt, or infection). If lid anatomy or function is abnormal, refer. If the condition is *not* idiopathic, for example if Sjögren’s syndrome or an unidentified underlying disease are suspected, refer.

**A3:** if SJS or OCP are suspected, refer urgently (within one week) to ophthalmologist

Possible management by Ophthalmologist

- drug treatment for underlying disease (e.g. SJS, OCP)
- ciclosporin eye drops (Ikervis)
- autologous serum eye drops
- electrolysis, cryotherapy
- protection with therapeutic contact lenses of all types
- permanent (surgical) occlusion of puncta
- tarsorrhaphy (surgical or botulinum toxin)
- transplantation of salivary gland/duct

Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see [http://gradeworkinggroup.org/toolbox/index.htm](http://gradeworkinggroup.org/toolbox/index.htm))

**Sources of evidence**


Dry Eye Disease (Keratoconjunctivitis Sicca, KCS)

| Pucker AD, Ng SM, Nichols JJ. Over the counter (OTC) artificial tear drops for dry eye syndrome. Cochrane Database Syst Rev. 2016;2:CD009729 |

LAY SUMMARY

Dry Eye Disease is also known by the medical term Keratoconjunctivitis Sicca, which means inflammation of the conjunctiva (the membrane overlying the white of the eye) and the cornea (the clear window of the eye) caused by dryness resulting from a deficiency or disorder of the tear film (the thin layer of tears covering the surface of the eye). It is a common condition affecting many people in the later decades of life. Most cases have no apparent cause but some are related to various inflammatory conditions, surgical treatment or as a side-effect of drug treatment. Some are caused by abnormalities of the eyelids or blinking, or by disorders of the Meibomian (oil) glands of the eyelid margin.

Patients complain of irritation of the eyes, a feeling that there is something in the eye, a discharge from the eye, and sometimes blurred vision. They notice that their symptoms are worse in windy or dry conditions or when irritants such as smoke are in the air. When they are examined in the clinic they may be found to have reduced tear production or increased tear evaporation (sometimes due to lack of normal oil gland secretion). There may be damage to the surface of the eye produced by the increased saltiness of the tears.

Tears can be supplemented with various drops and ointments. It is also possible to conserve natural tears by blocking the openings of the tear ducts, either temporarily with tiny plugs or permanently by surgery. Where the problem relates to a disorder of the oil glands, treatment is directed to the eyelids.
# Trauma (blunt)

**Aetiology**
- Blow to the eye: accidental (e.g. RTA, industrial, domestic, sports) or non-accidental (e.g. fist)
- Also known as ocular contusion

**Predisposing factors**
- Usually unexpected but may be vocational (e.g. boxing)

**Symptoms**
- Pain varies from mild to severe
- Epiphora
- Visual loss (variable)
- Photophobia
- Possible diplopia

**Signs**
- Mild cases (usually with good corrected vision)
  - eyelid swelling (oedema), ecchymosis (bruising)
  - conjunctival chemosis, subconjunctival haemorrhage
    - unexplained subconjunctival haemorrhages in babies and young children may indicate non-accidental injury
  - corneal abrasion
- Severe cases (usually with some loss of visual function)
  - infraorbital nerve anaesthesia (lower lid, cheek, side of nose, upper lip, teeth) may indicate orbital floor fracture
  - disturbance of ocular motility: restriction or diplopia due to tissue swelling or muscle tethering by orbital (‘blow-out’) fracture
  - enophthalmos (sunken eye) may also indicate orbital fracture
  - among paediatric patients, orbital floor blow-out fractures may occur with minimal soft-tissue signs (‘white-eyed blow-out fracture’)
  - nasal bleeding (direct trauma, or could indicate skull fracture)
  - corneal oedema or laceration
  - AC: hyphaema (blood in aqueous), uveitis, flare and cells
  - traumatic mydriasis
  - iridodialysis (tearing of iris from its attachment to ciliary body)
  - lens: evidence of subluxation, cataract, capsule damage
  - IOP may be increased secondary to obstruction of the trabecular meshwork by blood cells, inflammatory cells or pigment. IOP may be reduced because of scleral perforation (rupture of globe)
  - the likelihood of traumatic glaucoma following ocular contusion is increased where there is increased pigmentation of the trabecular meshwork, elevated baseline IOP, hyphaema, lens displacement and angle recession (widened angle recess)
  - vitreous haemorrhage
  - commotio retinae, retinal detachment or dialysis
  - traumatic macular hole
  - globe rupture (full thickness wound of eye wall)
  - relative afferent pupillary defect (indicates traumatic optic neuropathy)

**Differential diagnosis**
- Other causes of acute red eye
- Pre-septal cellulitis

**Management by Optometrist**
- Practitioners should recognise their limitations and where necessary seek further advice or refer
## Clinical Management Guidelines

### Trauma (Blunt)

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
<th>Evidence Base</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non pharmacological</td>
<td>Careful history required, including mechanism and time of injury.</td>
<td><em>GRADE</em>: Grading of Recommendations Assessment, Development and Evaluation (see <a href="http://gradeworkinggroup.org/toolbox/index.htm">http://gradeworkinggroup.org/toolbox/index.htm</a>)</td>
</tr>
<tr>
<td></td>
<td>Lid oedema: cold compress to ease swelling</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
<td></td>
</tr>
<tr>
<td>Pharmacological</td>
<td>Systemic analgesia e.g. paracetamol, aspirin, ibuprofen</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Non-steroidal anti-inflammatory drug (e.g. ibuprofen) where there is significant tissue swelling</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>In cases of corneal abrasion consider prophylactic topical antibiotic</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=weak)</td>
<td></td>
</tr>
<tr>
<td>Management Category</td>
<td>Management depends on severity of injury</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Mild cases:</strong> alleviation or palliation; referral unnecessary</td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Severe cases:</strong> first aid measures and emergency (same day) referral to A&amp;E</td>
<td></td>
</tr>
<tr>
<td>Possible management by Ophthalmologist</td>
<td>Assessment and investigation including imaging (e.g. X-ray, CT, MRI)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Treatment of globe rupture where present</td>
<td></td>
</tr>
<tr>
<td></td>
<td>May require hospital admission</td>
<td></td>
</tr>
</tbody>
</table>

**Evidence base**


**Sources of evidence**


**LAY SUMMARY**

The eye is well protected by the bony structures of the face that surround it (brow, cheek, nose) but it is sometimes injured by a direct blow, which is usually accidental but is sometimes the result of...
an assault. In mild cases this often results in bruising and swelling of the tissues around the eye (a 'black eye') which resolves fully in time leaving no after-effects; painkillers may be the only treatment needed. In more severe cases one or more of the bones of the orbit (the bony cavity in which the eyeball sits) may be fractured and this may cause the eye or one of the muscles that moves it to be displaced. The blow to the eye may also damage the structures inside the eye and may cause internal bleeding or raised eye pressure. Such cases need to be referred as emergencies to the ophthalmologist.
**Aetiology**

The incidence of chemical injuries to the eye has been reported to be 10.7 per 100,000 population per annum, representing an estimated 10% of ocular trauma treated in emergency departments. Most patients are males aged 16–25 years.

A wide variety of chemicals can be responsible for ocular injury, including:

**Alkalis**, such as:
- ammonia compounds (household cleaners, fertiliser)
- sodium hydroxide (drain and oven cleaners)
- lime, i.e. calcium hydroxide (cement, plaster)

(NB alkalis cause liquefactive necrosis and readily penetrate the eye)

**Acids**, such as:
- sulphuric (car batteries)
- hydrofluoric (glass etching)
- hydrochloric (>25% is corrosive)
- glacial acetic (wart, verruca treatment)
- citric (limescale removal)

(NB acids cause coagulative necrosis which impedes penetration of the eye)

**Detergents**, such as:
- free chlorine liberating compounds including sodium hypochlorite (bleach)

**Solvents**, such as:
- paint thinners
- petrol
- nail varnish remover

**Fixatives**, such as:
- formaldehyde
- glutaraldehyde

Contact lens solutions, including hydrogen peroxide; in clinics, tonometer disinfection fluids

Pepper spray, tear gases (CS, CN, CR)

Cyano-acrylate adhesive (superglue) – tube can be confused with eye drops or ointment

**Predisposing factors**

- Domestic accidents, DIY injuries
- Industrial injuries
- Assault
- Riot control
- Warfare

**Symptoms**

- Immediate pain, redness, epiphora
- Visual loss
- Severe chemical trauma may be relatively pain free (damage to superficial nerves)

**Signs**

- Burns to eyelids and surrounding skin
- Particulate matter under lid (evert to examine)
- Conjunctival chemosis and hyperaemia
- Limbal and conjunctival blanching (cessation of blood flow in superficial
### Trauma (chemical)

**Corneal epithelial defects ranging from superficial punctate keratitis through focal epithelial loss to sloughing of the entire epithelium**

- Raised IOP

**Corneal oedema and opacification in severe cases (may prevent view of anterior chamber, iris, lens or beyond)**

Various chemical trauma classification systems exist, e.g. those of Roper-Hall and Dua and the ILSI classification. Each of these establishes limbal ischaemia as dividing mild from more severe trauma.

### Differential diagnosis

- Corneal abrasion
- Other causes of acute red eye; history should aid the diagnosis

### Management by Optometrist

**Differential diagnosis**

- Corneal abrasion
- Other causes of acute red eye; history should aid the diagnosis

**Management by Optometrist**

**Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere**

#### Non pharmacological

**Immediate management**

- **irrigation should begin immediately at the scene of the accident with any non-toxic liquid (e.g. tap water)**
- **on presentation to clinical care, the patient should receive copious prolonged irrigation of the eyes with sterile normal saline (at least one litre); if not immediately available, use tap water**
- **irrigate for 15-30 min (with intermittent topical anaesthetic if required) or until pH between 7 and 8 (normal value 7.4, range 7.3 – 7.7): to measure, cease irrigation, wait for 1 min, apply universal indicator paper to fornix**
- **when pH normal, check again after additional 30 min**
- **remove any particulate matter, sweeping the fornices with a moistened cotton bud**
- **ascertain which chemical caused the injury**
- **check VA (important even if pain and/or swollen lids make this difficult)**
- **contact lens solution accidents: advise no contact lens wear until after satisfactory review**

(GRADE*: Level of evidence=moderate, Strength of recommendation=strong)

#### Pharmacological

- **In severe cases (i.e. where there is limbal ischaemia or loss of corneal transparency), no pharmacological intervention (immediate referral)**
- **In mild cases, e.g. contact lens solution accidents, give ocular lubricants (preferably unpreserved) for symptomatic relief**
- **For pain or photophobia, advise systemic analgesia and darkened room**

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

### Management Category

**In severe cases:**

- A2: first aid measures and immediate referral to A and E. Telephone the on-call ophthalmologist

**In mild cases:**

- B2: alleviation/palliation (normally no referral)

### Possible management by Ophthalmologist

- Further irrigation
- Admission to hospital where necessary
- Treatment with topical antibiotic, topical steroid, systemic ascorbic acid,
topical sodium citrate, systemic acetazolamide if IOP raised, other drugs
Surgical rehabilitation, e.g. amniotic membrane graft, limbal stem cell transplantation

### Evidence base

**GRADE: Grading of Recommendations Assessment, Development and Evaluation** (see [http://gradeworkinggroup.org/toolbox/index.htm](http://gradeworkinggroup.org/toolbox/index.htm))

**Sources of evidence**


### LAY SUMMARY

A variety of chemicals, coming into accidental or deliberate contact with the eye, can damage it. These include alkalis, acids, detergents, solvents, certain contact lens solutions and pepper spray or tear gas. Generally the damage is greatest with alkalis such as ammonia, found in some household cleaners, and sodium hydroxide, present in drain and oven cleaners, as these chemicals pass easily through the outer coat of the eye. Chemical injuries can occur in assaults, and in work, DIY or household accidents, also in riot control and warfare situations.

The result of a chemical injury to the eye is usually pain, redness and watering of the eye, all of which may be severe, and there may be loss of vision also. The task of the optometrist is to quickly judge the nature and extent of the injury and then, in all but mild cases, to flush the eye with large amounts of saline solution in an attempt to wash away the alkali or acid that may have caused the injury.

After this first aid, the optometrist will arrange for the ophthalmologist to see the patient as soon as possible on the same day. Depending on the severity of the injury, the ophthalmologist may admit the patient to hospital for further intensive treatment.
## Aetiology
Partial or full-thickness injury of outer wall of eye caused by sharp object
Common causes include: assault, industrial or work-related accident, DIY injury

## Predisposing factors
Male:female = 3:1

## Symptoms
- History of trauma
- Pain
- Visual loss

## Signs
<table>
<thead>
<tr>
<th>Lid laceration: assess depth, contamination and whether canaliculi involved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conjunctiva</td>
</tr>
<tr>
<td>• hyperaemia and chemosis</td>
</tr>
<tr>
<td>• look for foreign bodies</td>
</tr>
<tr>
<td>• assess depth of any conjunctival laceration</td>
</tr>
<tr>
<td>Corneal laceration</td>
</tr>
<tr>
<td>• check depth</td>
</tr>
<tr>
<td>• check for signs of perforation (shallow or flat AC, Seidel test +ve)</td>
</tr>
<tr>
<td>• possible iris damage (iridodialysis) ± iris prolapse into wound</td>
</tr>
<tr>
<td>Lens</td>
</tr>
<tr>
<td>• may be subluxated, dislocated, absent, or cataractous</td>
</tr>
<tr>
<td>Scleral laceration</td>
</tr>
<tr>
<td>Irregular pupil</td>
</tr>
<tr>
<td>Commotio retinae</td>
</tr>
<tr>
<td>Vitreous haemorrhage</td>
</tr>
</tbody>
</table>

## Differential diagnosis
- Non-penetrating (blunt) trauma
- Chemical trauma

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

### Non pharmacological
**DO NOT APPLANATE OR EXERT PRESSURE ON EYE**

- Take a careful history
  - patient’s description of events leading to trauma
  - nature of any known foreign body, its speed and size
  - check tetanus status
- If there is any suspicion of a full-thickness laceration of the globe
  - do not exert any pressure on the eye (including forcing the lids open)
  - advise patient not to cough or strain
- Check VA (important even if pain and swollen lids make that difficult)
- Protect eye by taping over it a rigid plastic shield (e.g. cartella)
- If penetrating object is still in the eye **do not be tempted to remove it**
- If iris protrudes from wound **do not attempt to push it back**
- Advise patient to take nil by mouth (except as below*)

*To assist swallowing of tablets, a small amount of water is permissible

### Pharmacological
Topical anaesthetic (to aid examination), systemic pain relief and anti-emetic as required

*To assist swallowing of tablets, a small amount of water is permissible

### Management Category
A2: first aid followed by immediate referral; no intervention. Telephone the on-call ophthalmologist
**Possible management by Ophthalmologist**

- Orbital X-ray, ultrasound, other investigations
- Surgical management of penetrating injury
- Prophylaxis of intra-ocular infection
- Follow-up includes examination for possible sympathetic ophthalmia affecting fellow eye (occurs in 0.1% of cases of penetrating trauma)

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*

**Sources of evidence**


---

**LAY SUMMARY**

Full or partial penetration of the outer coat of the eye (the clear part, the cornea or the white part, the sclera) can result from industrial, work-related or DIY injuries, or from assaults with sharp objects. Such injuries occur more commonly in males than females. Because they are so close to the eyeball, the eyelids may be injured also.

The optometrist will check the vision and examine the injured eye to discover the extent of the damage and whether there is full or partial penetration. Any foreign bodies will be noted but not removed. Evidence of damage to the internal structures of the eyeball, such as the lens of the eye, will be looked for.

The optometrist will prescribe or supply pain relief if necessary and make arrangements for the ophthalmologist to see the patient as soon as possible on the same day.

The ophthalmologist, having examined the patient, may arrange investigations such as X-rays or ultrasound and will decide on whether surgery is necessary, and if so how soon. In penetrating injury there is a very rare risk of inflammation in the other eye, a possibility that will be watched for as the patient is followed up.
### Aetiology

Inward misdirection of eyelashes towards the cornea, secondary to a number of conditions. These include distichiasis, in which an extra row of lashes grows from the Meibomian gland orifices.

- **Congenital**
  - Due to failure of epithelial germ cells to differentiate completely to Meibomian glands; autosomal dominant inheritance.

- **Acquired**
  - Entropion of any cause (see Clinical Management Guideline on Entropion).
  - Metaplasia of Meibomian glands leading to abnormal growth of lashes is usually secondary to severe chemical burn, Stevens-Johnson syndrome, ocular cicatricial pemphigoid, or chronic blepharoconjunctivitis.

### Predisposing factors

- Staphylococcal blepharitis
- Trachoma (a form of chronic conjunctivitis caused by *Chlamydia trachomatis*, which leads to tarsal conjunctival scarring, entropion and trichiasis)
- Cicatricial conditions (Stevens-Johnson syndrome, ocular cicatricial pemphigoid, chemical or mechanical trauma)
- Herpes zoster ophthalmicus

### Symptoms

- Ocular discomfort, irritation, foreign body sensation affecting one or both eyes
- *(NB: in the elderly and in diabetics, corneal sensitivity may be reduced)*
- Watery eye
- Red eye

### Signs

- Lash or lashes in contact with ocular surface
- Conjunctival injection
- Corneal epithelial abrasion
- Fluorescein staining of cornea and/or conjunctiva
- Long-standing complications:
  - pannus
  - corneal ulcer
  - infective keratitis

### Differential diagnosis

Other causes of ocular irritation / red eye

### Management by Optometrist

**Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.**

#### Non pharmacological

- **Epilation:** remove lash(es) with forceps. Advise patient that lash(es) will re-grow within 4-6 weeks, therefore epilation may need to be repeated *(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

- **If due to entropion, tape the eyelid for temporary relief of symptoms** *(GRADE*: Level of evidence=low, Strength of recommendation=strong)*

- **Consider therapeutic contact lens (silicone hydrogel soft, rigid limbal or rigid scleral)** for temporary relief of symptoms *(GRADE*: Level of evidence=low, Strength of recommendation=weak)*

#### Pharmacological

- **Ocular lubricants for symptomatic relief (drops for use during the day, unmedicated ointment for use at bedtime)** *(GRADE*: Level of evidence=low, Strength of recommendation=strong)*
NB Patients on long-term medication may develop sensitivity reactions which may be to active ingredients or to preservative systems (see Clinical Management Guideline on Conjunctivitis Medicamentosa). They should be switched to unpreserved preparations.

**Lid hygiene for associated blepharitis**

### Management Category

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild cases requiring epilation only:</td>
<td>B2: Alleviation/palliation: normally no referral</td>
<td></td>
</tr>
<tr>
<td>More severe cases requiring surgical intervention:</td>
<td>B1: Initial management (including drugs) followed by routine referral to ophthalmologist</td>
<td></td>
</tr>
</tbody>
</table>

### Possible management by Ophthalmologist

- **Electrolysis:** destruction of lash follicle by passing electric current into lash root. Suitable for single or small numbers of lashes. May require multiple treatments.
- **Cryotherapy:** nitrous oxide cryoprobe eliminates large numbers of lashes; may cause skin depigmentation.
- **Radiofrequency ablation**
- **Laser photoagulation:** repeated application of laser burns to the hair root and follicle. Various lasers have been used.
- **Treatment of predisposing ocular conditions**
- **Lid surgery if trichiasis secondary to entropion e.g. eyelid tightening in the form of evertting sutures and lateral tarsal strip. For trachoma trichiasis, full thickness tarsotomy and rotation of lash bearing tissue has been shown to be effective.**

### Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see [http://gradeworkinggroup.org/toolbox/index.htm](http://gradeworkinggroup.org/toolbox/index.htm))

**Sources of evidence**


### LAY SUMMARY

In trichiasis, the eyelashes point inwards towards the eye rather than outwards as normal. Rarely, the condition may run in families, so that babies may be born with it. Much more commonly it results from either entropion (turning in of the lower lid resulting from age-related slackness of the tissues) or long-standing inflammation of the eyelids which can cause abnormal eyelashes to grow inward.
out of the oil glands on the edge of the eyelid. In global terms the commonest cause of trichiasis is trachoma, an infection spread by flies and between people, which affects at least 150 million people worldwide. Trachoma occurs throughout the world, but especially among people in developing countries and in poor rural communities.

People with trichiasis usually have uncomfortable red eyes and a feeling of something in the eye. The optometrist will see that one or more lashes are in contact with the eye surface. This may cause damage to the eye surface, which can then become infected. The optometrist may decide to pull out the offending lashes, using fine forceps. However, the lashes will usually grow again within four to six weeks, so this is only a temporary solution. Sometimes a bandage contact lens is fitted to relieve the patient’s symptoms. If the problem persists, the optometrist will refer the patient to the ophthalmologist, who may decide to remove the lashes by electrolysis, cryotherapy (freezing treatment) or laser treatment. If the cause is entropion, a minor surgical operation may be all that is required.
## Aetiology

**Uveitis (anterior)**

Anterior uveitis (the most common form of uveitis: 75% of all cases of uveitis). Annual incidence 12 per 100,000 population

- iritis: inflammation predominantly affects iris
- iridocyclitis (more common): inflammation predominantly affects iris and anterior part of ciliary body (pars plicata)

The Standardisation of Uveitis Nomenclature (SUN) working group has developed an international standard for classifying uveitis:

- **Onset:** sudden or insidious
- **Duration:** limited, if it is ≤3 months, or persistent, i.e. >3 months in duration
- **Recurrent:** describes repeated episodes of uveitis separated by periods of inactivity without treatment of ≥3 months in duration
- **Chronic:** describes persistent uveitis characterized by prompt relapse (in <3 months) after discontinuation of therapy

### Endogenous aetiology

- majority of cases assumed to be autoimmune in origin and may be associated with systemic disease (e.g. Ankylosing Spondylitis, Behçet’s Disease, Juvenile Idiopathic Arthritis, Reactive Arthritis (also known as Reiter’s Syndrome), inflammatory bowel disease, Psoriatic Arthropathy, Sarcoidosis)
- prior infections (e.g. *Herpes simplex*, *Herpes zoster*, *Mycobacterium tuberculosis*, *Treponema pallidum* [the agent of syphilis], *Toxoplasma*)
- idiopathic (not associated with an underlying systemic disease)
- specific uveitis entities with distinct characteristics, e.g.:
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman syndrome
  - anterior segment ischaemia

### Exogenous aetiology

- external injury or infection

Anterior uveitis is traditionally classified as ‘non-granulomatous’ or ‘granulomatous’, based on the nature of the keratic precipitates

- non-granulomatous uveitis typically has an acute onset and shows fine KP. It is more likely to be idiopathic
- granulomatous uveitis typically presents as a chronic condition showing large, ‘mutton fat’ KP and iris nodules. It is more likely to be associated with systemic conditions

## Predisposing factors

Age over 20 years in 90% of cases (mean age at onset = 40 years)

Major histocompatibility complex antigen HLA-B27 is positive in 8% of the general population, but in approximately 50% of all patients with this condition.

Systemic disease as above: it is important to take a comprehensive medical history
### Symptoms
Onset usually sudden at first episode, gradual at subsequent episodes
Usually unilateral (if bilateral, more likely to be associated with systemic disease and more likely to become chronic)
- pain (dull/ache)
  - exacerbated on induced pupillary constriction (direct, near or consensual)
- photophobia
- redness
- decreased vision
- lacrimation

NB If condition recurrent, eye may be asymptomatic and white despite presence of inflammation

### Signs
- hyperaemia: circumcorneal (‘ciliary injection’)
- keratic precipitates (KP) – fine or ‘mutton fat’
- aqueous cells
- aqueous flare
- intraocular pressure commonly normal but raised in some cases
- posterior synechiae possibly causing pupil block and iris bombé
- iris nodules: Koeppel (small, near pupil), Bussaca (large, far from pupil)

Anterior vitreous cells may be seen in iridocyclitis but often will indicate intermediate ± posterior uveitis

Other signs include constricted or non-reactive pupil, cataract, chronic corneal oedema including bullous keratopathy

Dilated posterior segment examination is essential: check for cystoid macular oedema & posterior uveitis in both eyes

NB If condition recurrent, signs may be less apparent, and will vary according to severity and the specific underlying disease

### Differential diagnosis
- Glaucoma (acute angle closure)
- Other causes of acute red eye, e.g. endophthalmitis
- Lens-induced uveitis, intraocular foreign body

Other forms of uveitis
- intermediate uveitis: involves posterior ciliary body (pars plana), anterior choroid
- posterior uveitis: involves choroid ± retina, optic disc or retinal vasculature
- panuveitis: inflammation of the entire uveal tract

### Management by Optometrist
Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere

| Non pharmacological | Check intraocular pressure
Dilated fundus examination (both eyes) to exclude intermediate and/or posterior uveitis |
---|---|
| | Following the initiation of therapy: Monitor for ocular complications, including raised intraocular pressure |
| | Sunglasses for photophobia |
| | Spectacle near addition for cycloplegia |

GRADE*: Level of evidence=low, Strength of recommendation=strong
Patients should be instructed to return immediately if they experience deterioration of vision or increased pain

<table>
<thead>
<tr>
<th>Pharmacological</th>
<th>First episode:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Topical steroid: e.g. gutt. prednisolone acetate 1% or dexamethasone 0.1% hourly until eye is white or inflammation controlled</td>
</tr>
<tr>
<td></td>
<td>Topical cycloplegic (NB first check for possibility of angle closure): gutt. cyclopentolate 1% tds for up to 7 days</td>
</tr>
<tr>
<td></td>
<td>See within the first 3 days and if the uveitis is responding well to treatment reduce the topical steroid to every 2 hours for 5 days, then start tapering. The topical steroid should be tapered over not less than 6 weeks.</td>
</tr>
<tr>
<td></td>
<td>If no improvement at one week, refer to ophthalmologist</td>
</tr>
<tr>
<td></td>
<td>Second or subsequent episode:</td>
</tr>
<tr>
<td></td>
<td>Reinstate uveitic therapies as above</td>
</tr>
<tr>
<td></td>
<td>Refer to an ophthalmologist for systemic review and possible onward referral to rheumatologist</td>
</tr>
<tr>
<td></td>
<td>(GRADE*: Level of evidence=low, Strength of recommendation=strong)</td>
</tr>
</tbody>
</table>

**NB:** do not commence treatment if patient is known to have a history of corticosteroid-induced ocular hypertension or has had an episode of hypertensive uveitis

<table>
<thead>
<tr>
<th>Management Category</th>
<th>First episode:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>B2:</strong></td>
<td>Alleviation and palliation: normally no referral to ophthalmologist, where there is:</td>
</tr>
<tr>
<td></td>
<td>• non-granulomatous inflammation</td>
</tr>
<tr>
<td></td>
<td>• unilateral involvement</td>
</tr>
<tr>
<td></td>
<td>• no underlying systemic aetiology</td>
</tr>
<tr>
<td></td>
<td>• no posterior segment involvement</td>
</tr>
</tbody>
</table>

**A3:** Urgent (within one week) referral to ophthalmologist if:

| • no improvement after one week of pharmacological treatment |
| • granulomatous features from the outset |
| • hypopyon or fibrin in anterior chamber |
| • failure to break posterior synechiae |
| • bilateral disease |
| • posterior segment involvement |
| • inadequate pupil dilation (to exclude posterior uveitis) |
| • history suggestive of an underlying systemic aetiology |

**A1:** Emergency (same day) referral to ophthalmologist (no intervention) if:

| • significant reduction in vision |
| • severe pain |
| • significantly raised IOP |

**Second or subsequent episode:**

**B1 (modified):** pharmacological management followed by urgent referral
**Uveitis (anterior)**

### Possible management by Ophthalmologist

- May investigate aetiology of uveitis and possibly refer appropriately for further medical investigation (e.g. rheumatologist, clinical immunologist, infectious disease specialist)
- Treat secondary glaucoma
- Sub-Tenon's steroid injection may be required
- Possible sub-conjunctival injection of mydriatic
- Possible systemic immunosuppression

### Evidence base

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*

**Sources of evidence**

Huang JJ, Elia M. BMJ Best Practice: Uveitis. Mar 28; 2018
https://bestpractice.bmj.com/topics/en-gb/407


### LAY SUMMARY

The uvea is the coloured part of the eye, lying beneath the white part of the eye (the sclera) and comprising the iris (which gives the eye its blue, brown or other colour), the ciliary body (which controls focussing and secretes the clear fluid that fills the front of the eye) and the choroid (which nourishes the outer layers of the retina). Inflammation of the uvea is called uveitis. When this inflammation affects the iris and the ciliary body only, it is known as anterior uveitis. It is the most common form of uveitis and occurs in around 12 per 100,000 people per year.

Anterior uveitis may be caused by injury or infection, but the most common cause is inflammation elsewhere in the body.

Most patients are over the age of 20 years. Approximately half of them are born with an antigen known as Human Leucocyte Antigen (HLA) B27 and, as well as uveitis, this makes them more likely to develop inflammation elsewhere such as:

- psoriatic arthropathy (joint inflammation associated with an abnormal skin condition)
- ankylosing spondylitis (which affects the spine and its joints with the pelvis)
- inflammatory bowel disease (such as Crohn’s disease and ulcerative colitis), and
- reactive arthritis (joint inflammation as a reaction to infection of another part of the body)

The HLA-B27 antigen is found in only 8% of the general population.

Acute anterior uveitis usually comes on suddenly, producing a dull ache, redness, light intolerance and blurred vision in one eye. The optometrist sees evidence of inflammation in the anterior chamber (the front part of the eye). Using the slit lamp biomicroscope, he or she will see that the aqueous fluid, which fills the anterior chamber, contains tiny cells and protein, making it cloudy.
There may be nodules on the iris. Provided that there is no serious effect on vision, severe pain or raised eye pressure, or inflammation affecting other parts of the eye, the optometrist will usually prescribe steroid drops to control the inflammation, plus other drops to relax the pupil by widening it. If there is no improvement within one week, he or she will refer the patient urgently to the ophthalmologist. The ophthalmologist will prescribe other drugs as necessary to suppress the inflammation and to deal with other problems such as raised eye pressure.

If the uveitis recurs, investigations including blood tests and X-rays will usually be ordered to look for underlying inflammation elsewhere in the body. Controlling that inflammation may help to prevent further attacks of uveitis.
**Vernal Keratoconjunctivitis (Spring catarrh)**

| Aetiology | Rare allergic disorder of children (prevalence in Western Europe is 3.2 per 100,000 inhabitants) 
| More common in some other parts of the world, e.g. Mediterranean region, parts of Africa, Indian sub-continent 
| Complex immune reaction with raised IgE levels in the tears and serum, and mast cells and eosinophils in the conjunctival epithelium |

| Predisposing factors | Onset usually before 10 years of age; M:F = 3-4:1 
| Seasonal exacerbations (hence name) but condition may be active year-round if severe 
| Patients usually atopic with a history of eczema and asthma 
| Often a family history of atopic disease |

| Symptoms | Ocular itching 
| Watering 
| Mucoid stringy discharge 
| Blurred vision 
| Photophobia 
| Difficulty opening eyes on waking 
| NB: the symptoms are often asymmetrical in the two eyes |

| Signs | Stringy white mucous exudate 
| Palpebral, limbal and corneal manifestations: 
| **Palpebral** 
| • hyperaemia and chemosis of conjunctiva when active 
| • giant papillary hypertrophy (papillae 1mm or greater in diameter) of upper tarsus (‘cobblestone’ appearance) 
| **Limbal** 
| • hyperaemic, oedematous, thickened limbus 
| • Trantas’s Dots (discrete white superficial accumulations of eosinophils and degenerating epithelial cells) 
| **Corneal** (usually in upper third) 
| • punctate epithelial keratopathy 
| • macro-erosion (coalescent epithelial loss) 
| • plaque (deposited on Bowman’s layer, preventing re-epithelialisation); ‘shield ulcer’ in US terminology 
| • subepithelial scarring (often ring-shaped) 
| NB: the signs are often asymmetrical in the two eyes 
| These patients may also have keratoconus and/or atopic cataract |

| Differential diagnosis | Atopic keratoconjunctivitis (usually in adults; around puberty, VKC may metamorphose into this disease) |

| Management by Optometrist | Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere |

| Non pharmacological | Cold compresses may reduce acute symptoms (GRADE*: Level of evidence=low, Strength of recommendation=strong) |

| Pharmacological | Mast cell stabilisers e.g. gutt sodium cromoglicate 2% qds, gutt lodoxamide 0.1% qds 
| Because of the sight-threatening nature of this condition, and the frequent need for other medical specialist involvement, maintain low threshold for referral to the ophthalmologist (GRADE*: Level of evidence=moderate, Strength of recommendation=strong) |
**Management Category**

<table>
<thead>
<tr>
<th>Management Category</th>
<th>Mild cases (without active limbal or corneal involvement):</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>B2: Alleviation or palliation; no referral</strong></td>
</tr>
<tr>
<td></td>
<td>Initial management with mast cell stabilizers. VKC requires careful monitoring for sight-threatening complications</td>
</tr>
<tr>
<td></td>
<td><strong>B1: Possible prescription of drugs; routine referral</strong></td>
</tr>
<tr>
<td></td>
<td>Routine referral if mast cell stabilizers fail to provide symptomatic relief</td>
</tr>
</tbody>
</table>

**Possible management by Ophthalmologist**

- VKC often requires a multi-disciplinary approach (e.g. clinical immunologist, paediatrician). Other topical drugs used include steroids, immunosuppressants (e.g. ciclosporin, tacrolimus) and mucolytics (acetyl cysteine).
- Manual or laser surgery may be required for the removal of corneal plaque.

**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see [http://gradeworkinggroup.org/toolbox/index.htm](http://gradeworkinggroup.org/toolbox/index.htm))

Sources of evidence

LAY SUMMARY

Vernal Keratoconjunctivitis (VKC), also known as Spring Catarrh, is a rare but serious allergic disease affecting the eyes of young children, especially boys, who usually have other allergic diseases such as eczema and/or asthma. It usually begins before the age of 10 years and often disappears at puberty, though it may change at that time into another allergic eye disease known as Atopic Keratoconjunctivitis.

Children with VKC complain of itching of the eyes, watering and a stringy discharge. Their vision may be blurred and they may be excessively sensitive to light. One characteristic symptom is that they may have great difficulty in opening their eyes on awaking, and this and the very distracting effect of the condition may cause them to miss school.

VKC produces inflammation of the eye surface. On the underside of the upper eyelids, bumps shaped like tiny cobblestones appear. Substances released from this tissue can cause damage to the cornea (the clear window of the eye). Sometimes a whitish deposit, known as plaque, may accumulate on the cornea, which may also become scarred, causing problems with vision.

VKC is not a simple allergic condition like Seasonal Allergic Conjunctivitis (Hay Fever Conjunctivitis) as it involves various different types of immune reaction. This is why many cases need to be referred to the ophthalmologist for management, who in turn may refer them to specialists in other allergic diseases. Surgery is sometimes needed when plaque has accumulated on the cornea.
Aetiology

Pigmented lesions of the choroid are a relatively common finding in the course of a routine examination of the fundus. The overwhelming majority are choroidal naevi, which are benign with a low risk of malignant transformation. Congenital hypertrophy of the retinal pigment epithelium has distinctive ophthalmoscopic features. Choroidal melanoma is a rare and life-threatening melanocytic tumour that can sometimes be difficult to distinguish from naevus.

**Choroidal naevus**
- Area of increased choroidal pigmentation
  - approx. 20% within the macular region, 70% between macula and equator and 10% between equator and ora serrata
- Low malignant potential (<1%)
- Prevalence reported as 2-5% (based on large cross-sectional studies of multi-ethnic populations of US adults >40 years using retinal imaging). A higher prevalence (6.5%) was found in the Blue Mountains Eye Study (BMES) cohort of largely white Australians (99%), aged 49-97 years. Population studies in other ethnicities report a lower prevalence: Hispanics (2.7%), African Americans (0.6%).

**Congenital Hypertrophy of the Retinal Pigment Epithelium (CHRPE)**
- Benign congenital anomaly of the retinal pigment epithelium (RPE)
- Area of increased RPE hypertrophy and hyperpigmentation
- Malignant transformation is extremely rare, although documented enlargement in observed in 46% of those followed up photographically for >3 years.

**Uveal melanoma**
- 90% are choroidal, 6% arise from the ciliary body and 4% from the iris
- 1.3-8.6 cases per million per year in European-derived populations
- Life-threatening ocular malignancy (risk of metastatic disease)
  - detection and treatment at earliest stage improves prognosis
- Risk of metastases related to genetic tumour profile, mortality occurring almost exclusively in patients whose tumour shows chromosome 3 loss (monosomy). Greater tumour thickness indicates greater likelihood of lethal mutations and hence increased risk of metastasis
- Outcome poor once metastasis occurs; 1 year survival in 10-15%
- Mean age at presentation 60 years (but can occur at any age)

**Predisposing factors**

<table>
<thead>
<tr>
<th>Pigmented Fundus Lesions</th>
<th>Choroidal naevus</th>
<th>Choroidal melanoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevalence increases with age</td>
<td>Ethnicity: higher in whites than in blacks</td>
<td>Melanoma mostly arises de novo but can rarely develop from congenital ocular melanocytosis, ocular melanocytoma or</td>
</tr>
</tbody>
</table>
Pigmented Fundus Lesions

| naevus. Clinical features indicating malignancy include: symptoms, thickness > 2mm, presence of sub-retinal fluid, orange pigment (lipofuscin), proximity within 2 disc diameters of the optic disc, absence of overlying drusen and documented growth  
  - Risk factors for choroidal melanoma include: light coloured irides, fair skin, inability to tan, ocular congenital ocular melanocytosis (‘naevus of Ota’) (1:400 lifetime risk of uveal melanoma), and, rarely, neurofibromatosis  
  - The role of sunlight is uncertain, as most UV light is filtered by the lens |

| Symptoms | Choroidal naevi  
- Rarely symptomatic. Blurred or distorted vision can occur with sub-foveal naevi or when associated with sub-retinal fluid |

| CHRPE  
- Asymptomatic |

| Choroidal melanoma  
- Symptoms include: photopsia, blurred or distorted vision, floaters, or field loss. In 30% of patients, uveal melanoma is detected (usually by optometrists) before symptoms develop |

| Signs | Choroidal naevus  
- Typical naevi are small, flat and grey (though some are amelanotic), with a featureless surface  
- Atypical naevi are larger and dome-shaped, with or without drusen and/or traces of sub-retinal fluid  
- Drusen indicate chronicity so that their absence over a domed lesion is suspicious |

| CHRPE  
- Solitary, flat, well demarcated deeply-pigmented lesion (but may contain discrete non-pigmented areas called lacunae)  
- Elliptical or irregular  
- May show narrow non-pigmented line at margins of lesion  
- Patients with CHRPE may (very rarely) develop low-grade adenocarcinoma |

| Choroidal melanoma  
- Small choroidal melanomas are distinguished from naevi by:  
  - clumps of orange pigment (which are hyper-autofluorescent)  
  - subretinal fluid, most easily seen on OCT  
  - documented growth (which requires sequential colour photography)  
- Larger choroidal melanomas are dome-shaped, >2 mm in thickness, with serous retinal detachment. The mushroom (‘collarstud’) shape is almost pathognomonic for melanoma  
- About 5% of choroidal melanomas are diffuse (i.e. with a wide base but minimal thickening). These tumours are aggressive and often extend extra-ocularly by the time of diagnosis  
- Vitreous haemorrhage is rare, occurring only if the tumour has |
### Differential diagnosis

- Peripheral exudative haemorrhagic chorioretinopathy
- Circumscribed choroidal haemangioma
- Haemorrhagic detachment of RPE or retina
- Age-related macular degeneration
- Choroidal metastasis from tumour elsewhere

### Management by Optometrist

Practitioners should recognise their limitations and where necessary seek further advice or refer the patient elsewhere.

#### Non pharmacological

- Sequential colour photography is essential for demonstrating or excluding tumour growth
  - If imaging not available, make careful drawing with measurement
- OCT and autofluorescence imaging help to differentiate large naevi from small choroidal melanomas

(GRADE*: Level of evidence=low, Strength of recommendation=strong)

#### Pharmacological

None

#### Management Category

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
</table>
| A3 (modified) | Urgent (within one week) referral to ophthalmologist following telephone discussion  
- Choroidal Melanoma  
- ‘High-risk’ choroidal naevus  
  - documented increase in size, especially thickness  
  - symptoms  
  - sub-retinal fluid  
  - location within 2 disc diameters of optic disc  
  - clumps of orange pigment on the tumour surface |
| B2 (modified) | Regular surveillance; normally no referral to ophthalmologist  
- ‘Low-risk’ choroidal naevus  
- CHRPE |

#### Possible management by Ophthalmologist

**Choroidal naevus/CRPEH**
- Serial observation and imaging

**Choroidal melanoma**
- Managed by a supra-regional multidisciplinary team
- Pre-operative investigations include: ultrasonography, OCT, autofluorescence imaging and, if diagnosis uncertain, aspiration biopsy with a fine needle or vitreous cutter. Cytology confirms the diagnosis of melanoma, whereas genetic analysis (e.g. multiplex ligation-dependent probe amplification) determines whether the melanoma has metastatic potential

- Treatment dependent on staging and includes:
  - brachytherapy with a radioactive plaque  
  - proton beam radiotherapy  
  - laser therapy (usually as an adjunct to radiotherapy)  
  - surgical resection, trans-sclerally or trans-retinally  
  - enucleation

- Postoperative surveillance for metastases, according to genetic results
**Evidence base**

*GRADE: Grading of Recommendations Assessment, Development and Evaluation (see http://gradeworkinggroup.org/toolbox/index.htm)*

**Sources of evidence**


**Further information**

http://www.oculonco.com/
http://www.ocuarmelanomaonline.org
LAY SUMMARY

A Choroidal Naevus (similar to a mole on the skin) occurs inside the eye in the choroid, which is the layer of nourishing and supportive tissue between the retina (the part of the eye that receives light and sends images to the brain) and the sclera (the ‘white’ of the eye). Choroidal naevus cannot be seen from the outside and usually causes no symptoms, so that people who do not have professional eye tests may never know that they have this condition. Very rarely, a choroidal naevus transforms into a malignant melanoma. Certain features identify naevi with a high risk of transformation.

Choroidal naevus can be confused with a condition called Congenital Hypertrophy of the Retinal Pigment Epithelial (CHRPE), a harmless blemish, present from birth in some people.

A large choroidal naevus can be confused with Choroidal Malignant Melanoma, which threatens sight and requires urgent review by an ophthalmologist. Treatment of choroidal melanoma is aimed at preventing spread of the tumour to other parts of the body, if possible conserving the eye with useful vision. The ophthalmologist has several different treatment techniques available. Genetic testing of removed tumour material can show whether or not metastasis (spreading of the tumour to other parts of the body) is likely.

The optometrist who diagnoses a high-risk choroidal naevus or a choroidal melanoma is advised to refer the patient to the ophthalmologist urgently (within one week). Other cases can be monitored by the optometrist; however, such monitoring requires sequential photography to detect or exclude tumour growth, and a method for identifying patients who have missed their follow-up appointment and who need to be encouraged to undergo regular eye examination.