MODULE 10:10

Diagnosis and treatment of diseases affecting the anterior chamber (lens, iris, ciliary body and pupil)

Louise O'Toole

Anatomically, the anterior chamber is bounded in front by the cornea, laterally by the sclera and ciliary body, and posteriorly by the iris and lens. The angle of the anterior chamber contains the trabecular meshwork through which intraocular fluid drains. The angle recess begins just beyond the final iris roll. The floor of the recess is formed by the ciliary body which extends anteriorly to the scleral spur. On gonioscopy, the ciliary body is seen as a light brown structure. Anterior to the ciliary body is another narrower band, grey-white in colour which is the scleral spur. This represents the posterior boundary of the trabecular meshwork. The posterior portion of the trabecular meshwork overlies Schlemm’s canal and is the filtering portion of the meshwork. Schlemm’s canal is not normally visible but in traumatised eyes, especially when there is low intraocular pressure, blood can reflux back into the canal, producing a prominent pink line.

The trabecular meshwork is more prominent in some ocular conditions including pigment dispersion glaucoma, pseudoexfoliation syndrome and following acute angle closure glaucoma or blunt ocular trauma. The trabecular hyperpigmentation is caused by excessive shedding of pigment from the posterior surface of the iris. Peripheral anterior synechiae are abnormal adhesions of the iris to a portion of the anterior chamber angle. This is most commonly associated with chronic anterior chamber inflammation. Less commonly, they result from a flat chamber following intracocular surgery. They are also a feature following iris bombé. In essential iris atrophy, both of the progressive type and in Chandler’s syndrome (a rare disorder in which the epithelium proliferates, causing corneal swelling and glaucoma), the synechiae frequently extend onto the cornea. Peripheral anterior synechiae accompany ruberosis iridis and may complicate angle closure glaucoma.

Iridocorneal endothelial syndrome (ICE)

Iridocorneal endothelial syndrome (ICE) is a spectrum of diseases. There are three clinical categories: Chandler’s syndrome, Cogan-Reese syndrome, and essential iris atrophy. The unifying abnormality in all three entities is the abnormal, unilateral, hammered silver appearance of the corneal endothelium. This appearance is secondary to the presence of an abnormal corneal endothelial cell layer which has the capacity to migrate across the anterior chamber angle and onto the surface of the iris. Chandler’s syndrome presents with only the hammered silver appearance of the corneal endothelium. Cogan-Reese syndrome includes the corneal changes but also has either pigmented nodules on the iris or a diffuse naevus which covers the anterior iris. Essential iris atrophy presents with the corneal findings and corectopia (abnormal location of the pupil) with developments of stretch and melt holes with disease progression. Glaucoma is a feature of this syndrome as there is secondary synechial closure of the angle due to contraction of the abnormal tissue.

Iris and pupil

Congenital aniridia is a relatively rare condition in which the iris is extremely rudimentary. In a few patients, the visual acuity may be good, however, it is generally poor and seen in association with a manifest nystagmus. Absence of the foveal reflex along with disc hypoplasia and choroidal colobomas are characteristically seen in aniridia. There may be associated
corneal abnormalities – opacification, microcornea, sclerocornea as well as the presence of adhesions between the cornea and the lens. The main threat to sight from aniridia comes from the associated glaucoma which is present in 75% of cases and often from an early age. The glaucoma can be difficult to manage medically and patients may require tube shunting to adequately reduce the intraocular pressure. The gene defect for aniridia has been mapped to the short arm of chromosome 11. Both autosomal dominant and recessive modes of inheritance have been described. Miller’s syndrome is characterised by aniridia, Wilms’ tumour of the kidney, genitourinary abnormalities and mental retardation.

Axenfeld-Rieger’s syndrome consists of a spectrum of anterior segment dysgenesis (defective development). Axenfeld anomaly is characterised by a prominent and anteriorly displaced Schwalbe’s line. Strands of peripheral iris tissue project onto this line. In Rieger’s anomaly, Schwalbe’s line is posteriorly located and iridal anomalies include stromal hypoplasia, corectopia, pseudopolycoria (full-thickness colobomas of the iris which may appear to be multiple pupils) and ectropion uveae. Glaucoma is also a feature of Rieger’s anomaly. Rieger’s syndrome contains the ocular features of Rieger’s anomaly but in addition, dental and facial anomalies exist including few and small teeth, hypoplasia of the maxilla and hypertelorism.

A typical coloboma of the iris extends downwards or down and inwards from the pupil. Colobomas may also involve the ciliary body and continue posteriorly into the choroid. Colobomas form secondary to failure of closure of the optic cup during embryological development. Iris colobomas may be isolated or may form part of a syndrome. The CHARGE syndrome consists of Colobomas, Heart defects, choanal Atresia, Retarded growth, Genital and Ear abnormalities.

Lisch’s nodules are melanocytic hamartomas, usually clear yellow to brown, that appear as well defined, dome shaped elevations projecting from the surface of the iris (Figure 1).

Iris naevi tend to be flat or minimally elevated, densely pigmented lesions with blurred margins. Lisch’s nodules are the most common clinical finding in adults over 20 years of age with type I neurofibromatosis and are present in 95% of cases.

Brushfield’s spots tend to be multiple and are seen in patients with trisomy 21. They are white in colour and form a ring shape on the anterior surface of the iris. They represent condensations of iris stroma and are frequently associated with a hypoplasia of the peripheral iris. They are more distinct, more numerous and closer to the pupillary margin than the similar appearing opacities – Kruckmann-Wolfflin bodies seen in patients without trisomy 21. Other ocular associations with trisomy 21 include blepharitis and punctate cataracts.
albinism involves both the skin and eyes, whereas ocular albinism mainly affects the eyes with minimal to no skin involvement. Other ocular signs and symptoms of albinism include photophobia, refractive errors, monocular vision, strabismus, pendular nystagmus, foveal hypoplasia, and abnormal decussation of the optic nerve fibres.

Diffuse transillumination of the iris is seen in cases where there have been attacks of acute angle closure glaucoma with resultant iris ischaemia. Diffuse transillumination of the iris with a moth-eaten type of appearance is associated with diabetes mellitus.

Sectorial transillumination of the iris follows infection by herpes simplex or more commonly by the herpes zoster virus. It may be associated with iridoschisis or follow either cataract surgery or previous acute glaucoma. It is a feature of the iridocorneal-endothelial syndrome.

Peripupillary transillumination is a feature of pseudoxfoliation whereas the transillumination defects which are associated with pigment dispersion syndrome are located in the mid-periphery. Localised transillumination defects are seen following an iridectomy (surgical removal of a segment of iris). The iridectomy which is formed during trabeculectomy surgery is usually superior, whereas the iridectomy which is created during a vitrectomy with a silicone implant is situated inferiorly.

Iris neovascularisation is generally associated with ischaemic disease of the retina. The most common cause of retinal ischaemia is either a central retinal vein occlusion or advanced diabetic retinopathy. However, rubecous iridis is also seen in venous stasis retinopathy, following central artery occlusion, in association with malignant melanoma, or following chronic uveitis.

Heterochromia iridis refers to differences in iris colour. The differences are best seen in day light rather than at the slit lamp. The abnormal eye may be hypochromic or hyperchromic. Hypochromia may also be congenital. Congenital hypochromia iridis may be isolated or associated with systemic features such as
Waardenburg’s syndrome - hypertelorism, white forelock, white eyelashes, leukoderma and cochlear deafness. Congenital Horner’s syndrome is associated with reduced iridal pigmentation as sympathetic innervation is important for the migration of the melanosome.

Hypochromia is a feature of Fuchs’ heterochromic cyclitis. In the initial stages of Fuchs’ heterochromic cyclitis, the heterochromia may not be detectable in patients with a thick iris. Flattening of the anterior iris architecture is seen, resulting from loss of the anterior iris border layer and iris stroma, especially in the peripupillary area. It may also be difficult to see in very pale blue irides. Small, non-pigmented, translucent, star-shaped keratic precipitates (KPs) are numerous and are nearly pathognomonic; they have fine filament projections, and, unlike the KPs seen in most patients with uveitis, those in Fuchs’ heterochromic cyclitis are distributed over the entire posterior corneal surface. Although there may be anterior chamber activity, posterior synechiae are never present. Secondary glaucoma is a frequent complication as are posterior subcapsular cataracts.

Iris hyperchromia may be a congenital defect and may be associated with the naevus of Ota. This is a hamartoma of dermal melanocytes which may involve the skin, mucosal and ocular structures. In siderosis bulbi, there is a discolouration of the iris secondary to the breakdown of an intracocular iron foreign body. There follows diffusion of ferrous ions throughout the eye and the iris gradually assumes a rusty yellowish brown discolouration. There may be an associated mydriasis due to damage of the sphincter muscle. Cataract formation and retinal dysfunction are also seen. When a copper foreign body becomes embedded in the iris the resultant discolouration is green. The associated cataract is termed a “sunflower cataract”. A diffuse naevus or melanoma may simulate iris heterochromia. Hyperchromia may also be drug-induced – Latanoprost can cause hyperpigmentation of the iris and there may also be associated hypertrichosis of the eyelashes following chronic usage.

Anisocoria refers to a difference in pupil size. The pupils should be examined both in bright light and also in dim lighting conditions to determine which is the abnormal pupil. In bright light, an enlarged pupil fails to constrict whereas an abnormally miosed pupil fails to dilate in low lighting conditions. The accommodation reflex should also be checked in all patients presenting with pupillary abnormalities.

Anisocoria may be physiologically normal and occurs in 20% of the population. Both pupils react normally to light and accommodation. The interpupillary size is generally less than 2mm. The degree of anisocoria can vary from day to day and may even switch sides.

Horner’s syndrome produces miosis, anhydrosis (loss of sweating) and ptosis on the affected side (Figure 4). The amount of anisocoria should increase in dim versus bright illumination. This condition occurs secondary to interruption of the sympathetic supply to the eye. If Horner’s syndrome is congenital, there may be heterochromia of the iris.

The sympathetic innervation to the eye consists of a 3-neuron arc. First-order neurons descend from the ipsilateral hypothalamus through the brain stem and cervical cord to T1/T2. Here they synapse on ipsilateral preganglionic sympathetic fibres and travel to the sympathetic chain as second-order neurons to the superior cervical ganglion. At this location, they synapse on postganglionic sympathetic fibres. The third-order neurons travel via the internal carotid artery to the orbit and innervate the radial smooth muscle of the iris which serves to dilate the pupil. Postganglionic sympathetic fibres also innervate the muscle of Müller within the eyelid as well as the sweat glands of the face.

Horner’s syndrome is congenital, there may be heterochromia of the iris.
A lens may be colobomatous with notching seen in the inferior equator of the lens. The lens may also assume a posterior bulge termed posterior lenticonus which is associated with Lowe syndrome (involves multiple physical and mental problems). Anterior lenticonus is the term used to describe anterior bulging of the lens and this abnormality is a feature of Alport’s syndrome (which causes kidney failure). The lens may be pathologically small, termed microphakia or small and of spherical shape — termed microspherophakia.

The lens may become displaced from its normal anatomical location and this is termed ectopia lentis (Figure 6). If the lens is completely dislocated from its normal position it is said to be luxated, whereas if there is only partial displacement it is termed subluxated. The underlying abnormality of ectopia lentis is a dysfunction of the zonular fibres of the lens.

The most common cause of a dislocated lens is trauma. However, ectopia lentis may also occur spontaneously, either in isolation or in association with a systemic syndrome. Isolated ectopia lentis has an autosomal dominant inheritance with the genetic defect mapped to chromosome 15. Microspherophakia is an associated feature of this condition. Typically, the lens is displaced superotemporally.

The ectopia lentis et pupillae syndrome is characterised by asymmetric eccentric pupils that are displaced in the opposite direction of the dislocated lens. The condition is usually bilateral and typically autosomal recessive. The irides often appear atrophic with transillumination defects on slit lamp examination. Cataracts are also commonly seen in this condition.

Conditions associated with lens dislocation include Marfan’s syndrome, homocystinuria, Weil-Marchesani syndrome, hyperlysinaemia, sulphite oxidase deficiency, Ehlers-Danlos syndrome, and congenital cataract.
and Stickler syndrome. Marfan’s syndrome has an autosomal dominant inheritance. Its defect lies in a mutation in the fibrillin gene coded for on the short arm of chromosome 15. Features of Marfan’s syndrome include tall stature, arachnodactyly, increased joint laxity, mitral valve prolapse, aortic dilatation, axial myopia, and increased incidence of retinal detachment. Ectopia lentis is common, occurring in about 75% of patients. The dislocation is usually bilateral, symmetrical, and its direction is superotemporal.

Homocystinuria is an inborn error of metabolism most often caused by an absence of the enzyme that converts homocysteine to cystathionine. Affected patients typically have fair skin with coarse hair, osteoporosis, mental retardation, and poor circulation. Thromboembolic events constitute the major threat to survival for patients with homocystinuria and particularly so following general anesthesia. Luxation of the lens is common and tends to be bilateral, symmetrical and inferonasal.

Primary ocular disorders associated with ectopia lentis include congenital glaucoma/buphthalmos, pseudoexfoliation syndrome, syphilis/chronic uveitis, retinitis pigmentosa, megalocornea, aniridia, hypermature cataract and high myopia.

The main complication of ectopia lentis is optical distortion due to displacement and tilting of the lens. This may be managed by spectacle correction, however sometimes surgical excision of the cataract is required. The displaced lens may also cause an increase in intraocular pressure.

The clear crystalline lens reacts to disease by forming a cataract. Cataract development is also a normal response in the ageing process or may be congenital in origin (Figure 6). The World Health Organisation (WHO) has estimated that cataracts account for 16 million cases of blindness worldwide where blindness is a best corrected visual acuity of <3/60 in the better eye. In the United States, age-related lenticular changes have been reported in 42% of those between the ages of 52 to 64, 60% of those between the ages 65 and 74, and 91% of those between the ages of 75 and 85. The development of age-related cataract is multifactorial. There is an association between prolonged UV-B exposure and the development of cataracts. Smoking and drinking also increase the development of cataractous changes in the lens.

Age-related cataracts may be classified as cortical, nuclear, subcapsular or a combination of the three. A cortical cataract affects the anterior, posterior or equatorial cortex of the lens. The opacities originate as vacuoles and clefts between the lens fibres which then evolve into spoke-like radial opacities. A nuclear cataract forms secondary to an increase in the optical density of the lens nucleus. In childhood, the lens is colourless but through life, the lens nucleus gradually acquires a yellow hue so that in old age the lens may be brunescent (Figure 8). A subcapsular cataract may be anterior or posterior. An anterior subcapsular cataract forms secondary to fibrous metaplasia of the anterior epithelium of the lens. A posterior subcapsular cataract is associated with posterior migration of the epithelial cells of the lens. The Lens Opacities Classification System (LOCS) is useful in cataract cases as it grades the level of cataract against standard photographs. It defines the extent of opacification in the cortex and posterior and subcapsular zones and the intensity of opalescence in the nuclear zone.

The type of cataract that develops may be influenced by local changes in its environment. The area of the lens within the pupillary area has a higher exposure to aqueous humour and may be affected preferentially with the use of topical medications to the eye and also ultraviolet light. The equator of the lens is exposed to a high concentration of blood borne agents. Similarly, the posterior surface of the lens is influenced by pathology of the vitreous and retina.

**Traumatic cataract**

When there is severe blunt trauma to the eye, the lens responds to this contusional injury by forming a cataract. In a blunt injury, the lens may come in direct contact with the posterior surface of the iris. There is subsequent imprinting of iris pigment onto the anterior surface of the lens. This physical sign is termed a Yossius ring. The cataract typically has a stellate or rosette-like pattern and is subcapsular in position. It may appear quickly after the event or take some time to mature. As a consequence of either blunt or penetrating trauma, the capsule of the lens may be breached. If the capsular defect is small, the lens heals by forming a localised opacity. When the defect is large, this may result in total opacification of the lens.

Infrared energy/glass-blower’s cataract...
and electric shock are other rare causes of traumatic cataracts. Electric shock may also result in associated memory loss, deafness and optic atrophy. The cataract typically associated with electrical shock injuries is an anterior subcapsular opacity. Infrared energy characteristically causes a true exfoliation (or lamellar delamination) of the anterior lens capsule. Ionising radiation which is used to treat choroidal melanomas may cause secondary cataract formation as a side-effect.

**Presenile cataracts**

**Diabetes mellitus**
The degree of cataract formation in patients with diabetes mellitus is associated with the duration and the control of their disease. Diabetic cataracts have a similar appearance to age-related cataracts but they present at an earlier age in the diabetic patient. However, in the younger uncontrolled diabetic there may be a rapid onset of cataract within a few days. Here there is osmotic overhydration of the lens and subsequent development of myopia. The lens quickly becomes cataractous with dense, white anterior and posterior cortical subcapsular opacities. These are termed “snowflake cataracts” (Figure 9). Fine needle-shaped polychromatic cortical opacities may also form. If appropriate treatment is received by the patient, the rapid progression to a mature cataract may be arrested and a lamellar cataract will develop in its place.

**Myotonic dystrophy**
This is a chronic, slowly progressing, highly variable autosomal dominantly inherited multisystem disease. It is characterised by wasting of the muscles. Cataracts are a feature in over 90% of cases. The cataracts typically commence as polychromatic granules and flakes in the superficial cortex and then progress to form stellate posterior subcapsular opacities. The cataracts are often visually insignificant in the initial stages of formation.

Other signs of myotonic dystrophy include wasting of the temporalis muscles, ptosis, light-near dissociation, hypotony and a pigmentary retinopathy.

**Atopic dermatitis**
Approximately one tenth of patients with severe atopic dermatitis develop presenile cataracts before the age of 30. They are typically either a posterior subcapsular type cataract or a shield cataract. Presenile cataracts are also seen in the skin conditions of ichthyosis and pemphigus.

**Neurofibromatosis**
Type 2 neurofibromatosis is associated with the development of presenile posterior subcapsular or posterior cortical cataracts. Other ocular features of Type 2 neurofibromatosis include combined hamartomas of the retinal pigment epithelium and retina. Epiretinal membranes are also associated with this condition. Lisch’s nodules and cataracts develop in the second or third decade of life.

**Toxic cataracts**
Cataracts may develop secondary to the
topical or systemic use of some pharmaceutical agents. Both systemic and topical steroids are significant risk factors in the development of posterior subcapsular cataracts. Children are thought to be more susceptible to cataract formation compared to adults following the use of corticosteroids.

The cataracts associated with chlorpromazine rarely interfere with visual function. There is a deposition of fine yellow-brown granules on the anterior lens capsule. These may progress into large stellate opacities and finally anterior polar cataracts. Long acting cholinesterase inhibitors can cause tiny anterior subcapsular vacuoles.

Busulphan is an antimitotic drug used in cancer treatment and it may cause formation of a posterior subcapsular cataract. Amiodarone as well as causing corneal verticillata (vortex keratopathy) can be associated with the development of anterior subcapsular lens deposits. These innocuous cataracts are also a feature of patients with rheumatoid arthritis who receive treatment containing gold. The cataract associated with Wilson’s disease is termed a sunflower cataract.

Cataracts should be managed with refractive correction until the patient becomes symptomatic. Symptoms include a reduction in distance or near acuity or increased glare – typically when driving at night. It is then reasonable for the patient to undergo ocular surgery. Phacoemulsification is the current practice of lens removal and may be performed as a sutureless procedure under topical anaesthesia. The natural lens is removed and replaced with an intraocular lens calibrated to the patient’s refractive error. Like any ocular intervention there are risks of haemorrhage, infection and sympathetic ophthalmia, however these risks are low.

A few weeks to months after successful cataract surgery the patient may complain of a reduction in their visual acuity in their successfully operated eye. This may be due to the formation of a second cataract due to opacification of the posterior capsule (Figure 10).

This condition is treated by laser YAG posterior capsulotomy where a laser creates a hole in the fibrosed posterior capsule (Figure 11). Complications related to this procedure include retinal detachment, macular oedema, raised intraocular pressure and lens pitting. Lens pitting occurs when the laser is focused too anterior.

To avoid central lens pitting, the laser burns are best placed peripherally rather than centrally.

Conclusion
The anterior chamber of the eye shows the consequences of both ocular and systemic disease. By performing a thorough examination occult diagnoses may become apparent. The anterior chamber should be examined before pupillary dilation or important clinical signs will become obscured and missed.

About the author
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Figure 11
YAG capsulotomy

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Module questions

Please note, there is only one correct answer. Enter online or by form provided.

Course code: c-5846

An answer return form is included in this issue. It should be completed and returned to CET initiatives (c-5846) OT, Ten Alps plc, 9 Savoy Street, London WC2E 7HR by October 31 2007.

1. Iridocorneal endothelial syndrome (ICE) includes all of the following except:
a) Chandler’s syndrome
b) Cogan-Reese syndrome
c) Rieger’s syndrome
d) Essential iris atrophy

2. CHANGE syndrome components include all of the following except:
a) Coloboma
b) Heart defects
c) Renal defects
d) Ear defects

3. Features of Albinism include all of the following except:
a) sectorial transillumination of the iris
b) nystagmus
c) foveal hypoplasia
d) abnormal decussation of the optic nerve head fibres

4. Which one of the following is incorrect? An iridectomy:
a) is a surgically performed removal of a segment of iris
b) is performed by laser
c) when found in association with a trabeculectomy is usually situated superiorly
d) when found in association with a vitrectomy is usually situated inferiorly

5. Causes of ruberosis iridis include all of the following except:
a) central retinal vein occlusion
b) proliferative diabetic retinopathy
c) venous stasis retinopathy
d) retinal angiomatous proliferation

6. Which one of the following is incorrect? Fuchs’ hereditary cyclitis:
a) typically has star shaped keratic precipitates found both superiorly as well as inferiorly on the cornea
b) the affected eye is hyperchromic
c) posterior synchiae are not a feature
d) secondary cataracts may develop

7. Which one of the following is incorrect? In siderosis bulbi:
a) retinal dysfunction is a feature
b) iris discoloration is a feature
c) cataract formation is typical
d) the cornea has a Kayser-Fleischer ring

8. Which one of the following is incorrect? In anisocoria:
a) in a third nerve palsy, the abnormal pupil is dilated
b) in acute Holmes-Adies pupil, the abnormal pupil is dilated
c) in Horner’s syndrome, the abnormal pupil is dilated
d) following inadvertent instillation of atropine, the abnormal pupil is dilated

9. Which one of the following is incorrect? Holmes-Adies pupil:
a) is more commonly found in women
b) reacts to light but not to accommodation
c) is a common cause of anisocoria
d) may become bilateral

10. Which one of the following is incorrect? Ectopia lentis:
a) in Marfan’s syndrome, the dislocation is typically supertemporal
b) in homocystinuria, the dislocation is typically inferonasal
c) in associated with high myopia
d) is associated with high hypermetropia

11. Which one of the following is incorrect regarding cataracts?
a) Vossius ring may accompany a traumatic cataract
b) true exfoliation is associated with a glass blower’s cataract
c) the cataract associated with chronic steroid use is typically posterior subcapsular
d) a shield cataract is characteristic of neurofibromatosis

12. Which one of the following is incorrect regarding cataracts?
a) a sunflower cataract is seen in Wilson’s disease
b) most cataract surgery is performed under topical anaesthesia
c) a secondary cataract develops within a week following cataract surgery
d) there is a risk of endophthalmitis with cataract surgery

Please complete on-line by midnight on October 31 2007 - You will be unable to submit exams after this date – answers to the module will be published in our November 2 issue.

Recommended Reading

CET answers

These are the correct answers to Module 10 Part 9, which appeared in our September 7 issue

1. d is correct
The uvea is the pigmented part of the eye that lies between the cornea-sclera on the outside and the retina on the inside. It is made up of the iris, ciliary body and choroid.

2. c is correct
Anterior uveitis is the most common form of uveitis; accounting for around 75% of all cases.

3. c is correct
Anterior uveitis is a common disease that affects mainly adults, with a prevalence of 0.1%.

4. a is correct
HLA-B27 is a type of antigen (a molecule that stimulates an immune response), which is encoded for on part of chromosome 6.

5. a is correct
Some of the complications of anterior uveitis include an irregular, fixed pupil because of posterior synechiae, which are adhesions between the posterior surface of the iris and the anterior capsule of the lens, caused by the inflammation.

6. d is correct
Raised intraocular pressure, posterior synechiae and cataract are all complications of anterior uveitis.

7. c is correct
Three percent of all Emergency Department visits are ocular related, and conjunctivitis is responsible for approximately 30% of all eye complaints.

8. a is correct
Staphylococcus aureus, Streptococcus pneumoniae, and Haemophilus influenzae are common causes of bacterial conjunctivitis.

9. c is correct
A variety of viruses can be responsible for conjunctival infection; however, adenovirus is the most common cause.

10. a is correct
The symptoms the patient experiences are of ocular itching, redness, burning, and tearing.

11. b is correct
Atopic keratoconjunctivitis is associated with a 95% prevalence of concomitant eczema and an 87% prevalence of asthma.

12. b is correct
Giant papillary conjunctivitis is a common complication of contact lens wear.

An ever increasing number of practices are relying on The Outside Clinic to solve their patient requests for domiciliary eye testing. Under the Code of Ethics and Guidelines for Professional Conduct - Aug 06, it states: "If an Optometrist does not offer a domiciliary service, information should be available in practice as to how a patient, carer, General Medical Practitioner (GMP) or others may access such services provided by other optometrists."