Managing the Red Eye

Speaker Notes

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A GUIDE TO PRESENTING
Managing the Red Eye

Managing the Red Eye introduces the primary care physician to skills useful in evaluating the red eye and provides a practical clinical approach to diagnosis and treatment of many common red eye disorders. Additionally, the audience will learn how to recognize more serious, vision-threatening red eye disorders for prompt referral to an ophthalmologist.

The program takes an anatomic approach to common red eye disorders and their management. Normal anatomy is reviewed as it relates to the pathophysiology of common diseases that contribute to the red eye. Included are disorders of the ocular adnexa (lids, orbit), lacrimal system, ocular surface (conjunctiva and sclera), and anterior segment (cornea and anterior chamber). Key concepts, such as side effects of topical steroids, and management for such emergent red-eye conditions as cellulitis, chemical burns, and acute angle-closure glaucoma are presented.

Approximate Running Time

50 to 90 minutes

Suggested Audience

- Family physicians
- Emergency physicians
- Internists
- Pediatricians
- Medical students, interns, residents
- Emergency-room personnel (non-MD)
- State and local meetings of national medical societies, AAFP, AAP, ACP, ACEP
INTRODUCTION

Physicians frequently encounter patients who complain of a red eye. This slide program provides an approach to differential diagnosis of conditions that can cause a red eye, helping physicians understand which conditions causing a red eye require immediate treatment, which may wait a few days, and which do not require treatment.

SLIDE 1

All subjective ocular complaints fall into three categories of symptoms: decreased vision, pain, and redness. Different types of decreased vision can be blurred vision from a foreign body disrupting the corneal surface or a dark haze that might come from a hyphema. With a careful history, different kinds of pain can be elicited, such as photophobia from corneal edema resulting from angle closure glaucoma or uveitis, foreign body sensation from an abrasion, or deep boring pain from scleritis or severe uveitis. Careful examination will show if the redness is unilateral, localized, or involves the lids. Eyelid disorders frequently bring patients with a red eye to the doctor, and so the lids should be considered as part of a systematic evaluation of ocular complaints.

Redness of the eyes and lids is caused by three types of problems: Mechanical trauma such as a foreign body or abrasion, chemical trauma such as an acid or alkali burn, and infection/inflammation, such as a corneal ulcer or uveitis.

SLIDE 2

SLIDE 3

DIFFERENTIATE RED EYE DISORDERS

- Needs immediate treatment
- Needs treatment within a few days
- Does not require treatment

SUBJECTIVE EYE COMPLAINTS

- Decreased vision
- Pain
- Redness

Characterize the complaint through history and exam.

TYPES OF RED EYE DISORDERS

- Mechanical trauma
- Chemical trauma
- Inflammation/infection
In order of urgency, the conditions that cause most red eye complaints are (1) chemical injury, (2) angle-closure glaucoma, (3) ocular foreign body, (4) corneal abrasion, (5) uveitis, (6) conjunctivitis, (7) ocular surface disease, and (8) subconjunctival hemorrhage. There may be different subsets in each category; for instance, conjunctivitis may be bacterial, viral, or allergic. Ocular surface disease may be an ectropion causing exposure, dry eyes, or an inflamed pinguecula. Some entities may cross categories: an infection early on may be bacterial conjunctivitis with minimal discomfort, but if it becomes a corneal ulcer it will initially feel like a foreign body. If it advances to endophthalmitis, it will cause severe uveitis-like pain.

**EVALUATION**

A systematic diagnostic approach to the patient with a red eye will help the physician reach a differential diagnosis that will include most of the causes of a red eye. As with any diagnostic problem, the information obtained from a careful history and examination should direct the approach to management. The “redness” in a red eye usually comes from dilated conjunctival blood vessels (the sclera is less vascular), as in the case of “pink eye,” or, rarely, torn blood vessels, which may exude bright red blood in a subconjunctival hemorrhage. The onset of a red eye, duration, and clinical course should be recorded to help distinguish the causative agent: trauma, chemicals, infection, allergy, or systemic conditions.
Specific symptoms may reveal the cause of the red eye. For example, itching typically signifies allergies. A burning sensation suggests lid, conjunctival, or tear film disorders, or corneal abrasions or foreign bodies. A foreign-body sensation might signify an embedded foreign body, a corneal abrasion, or an inturned eyelash. Localized lid pain or tenderness in the lids is a common presenting complaint of a hordeolum or an acute chalazion.

Deep, intense, aching pain that is not localized may reflect a large corneal abrasion, scleritis, iritis or acute glaucoma. Photophobia, pain when exposed to bright light, is caused by ciliary body muscle spasm, and indicates problems arising from the anterior segment of the eye, such as corneal abrasions, iritis, and acute glaucoma. A halo seen around a light is caused by corneal edema, seen in acute glaucoma and uveitis. Halo vision without pain can also be seen in contact lens overwear and cataracts.

To evaluate the red eye, the primary care physician needs a visual acuity chart, a penlight with a blue filter, fluorescein dye, and topical anesthetic drops.
The examination should begin with a visual acuity recording. A Snellen chart at 20 feet should be available in most offices, but a near vision card can be used. For young children, an eye chart using pictures can be used. Patients who wear eyeglasses or contact lenses should wear them for testing if possible. Remember that a patient over 40 years of age with good distance vision probably still needs reading glasses for near vision.

A red eye with decreased vision could signal a vision-threatening disorder. In general, red eyes with no vision loss can usually be treated by family physicians, but red eyes with any vision compromise should be referred where possible to an ophthalmologist.

After visual acuity is checked, systematic examination of the eye and adnexa should then be conducted, starting anteriorly with the face and lids and moving posteriorly to the globe. The face, orbital area, and lids are inspected first, then the ocular movements, and finally the globe itself. A slit-lamp biomicroscope is essential for examination of the anterior chamber, although careful scrutiny of the cornea with a penlight can yield a wealth of information.

Similarly, a tonometer (Schiotz, applanation, or TonoPen) to check intraocular pressure is the easiest way to rule out angle closure glaucoma, but a careful history and penlight exam can elicit the possibility of that condition.
A number of conditions cause redness of the ocular adnexa, although they may not actually cause the eye itself to become red. These conditions are discussed here because many lid problems are intricately connected to ocular surface disease and infections. Most of these conditions can be easily diagnosed and managed in the office. A cross-sectional view of the normal eyelid demonstrates anatomy pertinent to these disease entities. Anteriorly, note the skin, muscle, eyelashes, and perifollicular glands.

Hordeolum and Chalazion

Surrounding the follicles at the base of the eyelashes are oil glands, which, when obstructed, produce a hordeolum, or stye. A hordeolum may look like a pimple and develops near the skin surface on the anterior margin of the lid, adjacent to the cilia. Hordeola with swelling only are usually not infected, although redness and discomfort may be signs of infection.

The meibomian gland is a sebaceous gland that secretes the oily component of tears. There are approximately 30 to 40 vertically oriented meibomian glands across a normal lid. The meibomian glands are in the posterior aspect of the lid, behind the orbital septum and just in front of the cartilage tarsal plate, which provides support for the lids.
The meibomian glands drain through small opening on the posterior edge of the lid margin. When obstructed, these glands may produce a tender, red swelling in the adjacent lid tissue called a chalazion.

Treatment of a hordeolum or chalazion is aimed at promoting drainage of these inflamed glands. Hot compresses (warmer than lukewarm but not so hot that they burn) applied to the affected lid area externally for 10 minutes, 3 times daily, are highly effective for acute or subacute lesions. Compresses may have to be continued for several weeks until the condition is resolved. Because both conditions are usually sterile, topical antibiotics are usually unnecessary. Should a chalazion become a chronic, nontender, localized mass, drainage is achieved by incision and curettage by an ophthalmologist. Systemic antibiotics are usually not indicated for these localized lid disorders unless diffuse cellulitis also is present.
Blepharitis

Blepharitis is a chronic eyelid inflammation affecting the eyelashes and the glands surrounding the eyelashes, and sometimes associated with dry eyes. Seborrhea is noted as collarettes of dried skin and wax around the base of the lashes of the upper and lower lids. Associated localized redness may be caused by Staphylococcal infection. Typically, a patient complains of burning, mattering of the lashes, and eyelids sticking together upon awakening, but patients also may be asymptomatic.

This slide shows collarettes of dried skin and wax at the base of the eyelashes in a patient with blepharitis. Frequently seborrhea of the scalp, eyebrows, ears, and face is noticeable, and rosacea of the face may be present.

DISORDERS OF THE OCULAR ADENE

BLEPHARITIS

- Inflammation of lid margin
- Associated with dry eyes
- Seborrhea causes dried skin and wax on base of lashes
- May have Staphylococcal infection
- Symptoms: lid burning, lash mattering

Collarettes on eyelashes of patient with blepharitis
Treatment of blepharitis is directed toward proper face and lid hygiene. Instruct the patient to use hot compresses to loosen the crusting and to cleanse the lashes twice daily with a washcloth, cotton-tipped swabs moistened with nonirritating shampoo (such as a baby shampoo) diluted with water, or commercially available over-the-counter eye scrub pads. Other treatment options include applying antibiotic ointment, such as erythromycin, to the lids, or applying an antibiotic-steroid ointment, such as Tobradex or Blephamide. The combination antibiotic-steroid ointments can reduce inflammation in conjunction with other treatments. Oral doxycycline (Vibramycin 100 mg daily for 1 month) is helpful in treating refractory cases by changing the nature of the secretions produced by the meibomian glands.

Cellulitis

Cellulitis anterior to the orbital septum presents as edema and erythema of the lids. The lids are often tender to the touch. The edema may be so severe that the lids are swollen shut. In cases of anterior (periorbital or preseptal) cellulitis, the visual acuity, pupils, and mobility are normal, and there is no proptosis. These cases should be treated with systemic antibiotics and warm compresses. A CT scan should be considered if there are concerns that the orbit is involved or if the condition fails to respond promptly to antibiotic therapy.
If the cellulitis extends posterior to the orbital septum, a true medical emergency exists. Because of the vision- and life-threatening potential of orbital cellulitis, physicians should be aware of the clinical manifestations of this condition. Treatment should be started as soon as possible and consultation with an ophthalmologist should be obtained promptly.

The signs of orbital cellulitis include red and swollen lids and conjunctiva, as seen in the top photograph. Characteristically, ocular motility is impaired and there is pain on eye movement, as seen in the bottom photograph. Because the infection is posterior, the periorbital area may seem relatively uninflamed. The eye may protrude forward because of orbital swelling due to inflammation (proptosis), as seen in the middle photograph. Often the patient with orbital cellulitis will have fever and leukocytosis. Optic nerve involvement is signaled by decreased vision, an afferent pupillary defect, and optic disc edema. Meningitis can result from spreading along the optic nerve.

Management of orbital cellulitis should include hospitalization with immediate ophthalmology consultation. An evaluation for infection should include blood culture. Diagnosis can be assisted with an MRI or CT scan of the orbits. If pre-existing sinus disease (frequently associated with orbital cellulites) is present, an ENT consult is indicated.
Initiation of treatment with IV antibiotics is urgent and should result in improvement within 24 hours. Specific antibiotics should be chosen as clinically indicated. The most common causative agents of orbital cellulitis are *Staphylococcus aureus*, *Streptococcus* species, and *Haemophilus influenzae*. Diabetic, chronically ill, or immunologically suppressed patients may harbor a rapidly progressive fungal infection, and surgical debridement may be indicated in these cases. Surgery may also be necessary if there is no rapid response to IV antibiotics, or if the MRI or CT scan reveals a subperiosteal abscess. Complications of orbital cellulitis include cavernous sinus thromboses and meningitis.

**LACRIMAL SYSTEM DISORDERS**

Another red-eye condition commonly presenting to the primary care physician arises from abnormalities of the tear drainage system. Under normal conditions, tears are produced by the lacrimal gland and drain into the nose by way of lacrimal drainage structures: the puncta, canaliculi, common canaliculus, lacrimal sac, and nasolacrimal duct.
Nasolacrimal Duct Obstruction

Congenital or acquired obstruction of the nasolacrimal duct produces a characteristic clinical picture of a persistent tearing and occasionally discharge that fails to respond completely to topical antibiotics. A swollen, inflamed lacrimal sac (shown), termed dacryocystitis, may develop. Because secondary infections may arise from a blockage of outflow, definitive treatment depends on relieving the obstruction.

Nasolacrimal duct obstruction in the congenital form arises from persistent congenital membranes in the nasolacrimal duct that block the outflow of tears. In such cases, the parent should be taught to compress or massage the lacrimal sac once a day in an attempt to force the contents of the swollen lacrimal sac through distal obstructive membranes and into the nose. Approximately 90% of congenital obstructions will resolve spontaneously by 12 months of age but will only rarely resolve after that age. If tearing and chronic discharge persist beyond 6 to 8 months, the patient should be referred to an ophthalmologist for probing and irrigation of the nasolacrimal duct, which is usually done at 12 months of age. A single probing is curative in the majority of cases. Systemic antibiotics are indicated if dacryocystitis develops.
The most common causes of adult acquired nasolacrimal duct obstruction are trauma and recurrent infection of the lacrimal sac, causing stenosis and scarring. If secondary dacryocystitis is present, systemic antibiotics should be administered during the acute phase. Surgical intervention may be indicated after one episode of dacryocystitis. A surgical procedure to create a fistula between the lacrimal sac and the nose is necessary for recurrent or chronic cases, which rarely respond to medical therapy alone; however, surgery is usually curative.

**OCULAR SURFACE DISORDERS**

**Conjunctival/Scleral Anatomy**

In the normal eye, the conjunctiva forms a smooth, moist lining for the eyelids (the palpebral conjunctiva) and the anterior part of the eyeball (the bulbar conjunctiva). It is transparent tissue containing small blood vessels.
Conjunctivitis

When inflamed, both the bulbar and palpebral conjunctival blood vessels become dilated and readily apparent. This contrast is particularly evident if the blood vessels break and bleed. Red eyes attract attention, leading the patient to seek medical advice. A thorough clinical history and examination will allow the primary care physician to establish a diagnosis and treatment plan. An ophthalmologist should be consulted if an infection is suspected and vision is impaired, or the patient fails to respond to therapy in 3 to 4 days.

The major causes of primary conjunctivitis in adults are bacteria, viruses, and allergies. A knowledge of the symptoms—itching, for example, is characteristic of allergies—will assist in making a diagnosis.

The nature of the discharge, if any, can be helpful in determining the origin of the conjunctivitis. Purulence suggests bacteria; watery, serous discharge is associated with viruses, and watery discharge with stringy, white mucus is characteristic of allergies. Itching is often diagnostic for allergic conjunctivitis. In all cases of red eye, palpate for preauricular lymph nodes, a frequent finding in contagious viral conjunctivitis.

**ADULT CONJUNCTIVITIS: MAJOR CAUSES**
- Bacterial
- Viral
- Allergic

**CONJUNCTIVITIS: DISCHARGE**

<table>
<thead>
<tr>
<th>Discharge</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Purulent</td>
<td>Bacterial</td>
</tr>
<tr>
<td>Clear</td>
<td>Viral*</td>
</tr>
<tr>
<td>Watery, with stringy: white mucus</td>
<td>Allergic**</td>
</tr>
</tbody>
</table>

* Preauricular lymphadenopathy signals viral infection
** Itching often accompanies
**Bacterial Conjunctivitis**

*Staphylococcus* species, usually harbored in the skin, are the most common cause of conjunctivitis. *Streptococcus* and *Haemophilus* species, harbored in the respiratory system, are the next most common. Bacteria frequently cause a secondary purulent infection in patients with viral conjunctivitis. All common bacteria may cause conjunctivitis.

In the presence of a mild purulent discharge with a clear cornea, the primary care physician may begin treatment. Topical ophthalmic antibiotic solutions, applied 4 times daily, should be prescribed for 7 days. Bacterial conjunctivitis is treated with a broad-spectrum topical antibiotic such as, erythromycin, sulfacetamide, trimethoprim-polymyxin, an aminoglycoside, or a fluoroquinolone. Warm compresses applied several times a day should be included in the treatment regimen. If there is no significant clinical improvement in 3 days, referral to an ophthalmologist is in order.

In patients with a copious purulent discharge, *Neisseria gonorrhoeae* should be suspected. A conjunctival swab for stat Gram’s stain and culture are in order. Referral to an ophthalmologist is very important because corneal involvement may develop and perforation is possible.
**Viral Conjunctivitis**

In contrast to bacterial conjunctivitis, viral conjunctivitis produces a discharge that is usually watery. The most common cause of viral conjunctivitis by far is adenovirus.

Viral conjunctivitis is highly contagious, and hand washing is very important to avoid infection. Infected hospital personnel, daycare workers, food handlers, and those in similar occupations should avoid contact with others. This may necessitate taking up to 2 weeks’ time off from work. Palpable preauricular lymph nodes are an important sign differentiating viral from bacterial conjunctivitis. The patient may have an upper respiratory infection, a sore throat, fever, and generalized malaise, or someone in the family may have had these symptoms.

Viral conjunctivitis is a self-limited entity, and no specific treatment is generally indicated. However, if the patient has discomfort or moderate conjunctival chemosis, the patient should be referred to an ophthalmologist. Unfortunately, the condition may last for weeks, although most cases of viral conjunctivitis resolve in 14 to 21 days. If the conjunctivitis or symptoms persist beyond 2 weeks or there is pain, photophobia, or decreased vision, the patient should be referred to an ophthalmologist.
**Allergic Conjunctivitis**

Allergic conjunctivitis is characterized by lid or conjunctival edema often associated with a watery discharge and a white, stringy mucus. Itching is the predominant symptom and is sometimes accompanied by burning. The tarsal conjunctiva has a velvety appearance from papillary hypertrophy, and conjunctival chemosis is present.

Allergic conjunctivitis frequently occurs in patients with hay fever, asthma, or eczema. Contact allergy is associated with drugs, chemicals, or cosmetics contacting the conjunctiva or eyelids. The offending drug or allergen should be eliminated. Most allergic conditions can be treated symptomatically with topical antihistamines or artificial tears. Patients refractory to local forms of treatment should be referred to an ophthalmologist.

**Neonatal Conjunctivitis**

Neonatal conjunctivitis is an inflammation of the conjunctiva that occurs during the first 4 weeks of life. The timing of the conjunctivitis may be helpful eliciting the etiology. Possible causes include bacteria, such as *Neisseria gonorrhoeae*, *Staphylococcus*, and *Streptococcus*; *Chlamydia*; and viruses, such as herpes. Because some these causes of neonatal conjunctivitis have serious systemic manifestations as well as ocular manifestations, precise diagnosis and treatment are important.

**ALLERGIC CONJUNCTIVITIS**

- Associated conditions: hay fever, asthma, eczema
- Contact allergy: chemicals, cosmetics, pollen
- Treatment: topical antihistamine/decongestant drops
- Systemic antihistamines if necessary for systemic disease
  
  Refer refractory cases.

**NEONATAL CONJUNCTIVITIS: CAUSES**

- Bacteria (*N. gonorrhoeae*, 2–4 days)
- Bacteria (*Staphylococcus, Streptococcus*, 3–5 days)
- *Chlamydia* (5–12 days)
- Viruses (eg, herpes, from mother)
The infant with gonococcal conjunctivitis presents with swollen lids, heavy purulent exudate, “beefy-red” conjunctiva, and conjunctival edema. The gonococcal organism can penetrate the intact corneal epithelium, producing corneal ulceration and perforation if treatment is delayed. When gonococcal conjunctivitis is suspected, referral to an ophthalmologist is urgent. A combination of local and systemic therapy will be necessary.

Chlamydial infections are a leading cause of neonatal conjunctivitis because of the number of infants exposed to *Chlamydia* during vaginal delivery in infected mothers. The typical picture is a mild, unilateral or bilateral, mucopurulent conjunctivitis, with moderate lid edema and infection. Pneumonitis and otitis media often accompany those ocular findings. Cultures and smears are required to make this diagnosis because it may be impossible to clinically differentiate these conditions from neonatal bacterial conjunctivitis.

Chlamydial conjunctivitis is treated with erythromycin ointment, applied 4 times daily for 4 weeks. Oral erythromycin, 40 to 50 mg/kg/day in 4 divided doses, should be given for 2 to 3 weeks. In addition, both parents should be examined and treated as appropriate.
Subconjunctival Hemorrhage

This red eye is secondary to subconjunctival hemorrhage. A patient with this condition typically presents with a bright red eye, normal vision, and no pain. Patients may be on anticoagulation, aspirin, or high doses of vitamin E. In some cases, the hemorrhage is preceded by coughing or straining. There is no therapy except reassuring the patient that the condition is not serious, vision is not threatened, and that the blood will clear in 2 to 3 weeks.

Hematologic coagulation studies are usually not indicated unless there are associated retinal hemorrhages. A careful history should confirm that the hemorrhage was not associated with trauma, or that the subconjunctival hemorrhage might conceal the entrance wound of a small perforating foreign body.

Dry Eyes

Tears, because of their lubricating and bacteriostatic properties, are essential for the maintenance of a healthy cornea and conjunctiva. A deficiency in tear production may result in a dry eye, also known as keratoconjunctivitis sicca, a relatively common condition that may be managed by the primary care physician with frequent instillation of artificial tears.

Patients with severe dry eyes who are not relieved with artificial tears several times a day should be referred for evaluation and possible treatment with intensive use of nonpreserved artificial tears, punctal occlusion, or cyclosporine drops (Restasis).
Symptoms of the dry eye include burning and a foreign-body or “gritty” sensation. Paradoxically, discomfort from dry eyes may stimulate reflex tearing from the lacrimal gland. Symptoms usually exceed the signs of this common condition. Symptoms are made worse by activities that require concentration and subsequently reduce the normal blink reflex of every 3 to 4 seconds, such as reading, using a computer, watching television, or driving. Long airplane flights also cause excessive drying from low humidity.

Dry eyes tend to get worse with advancing age. Tear deficiency states can also be seen in a number of other conditions, such as rheumatoid arthritis. Some conditions that result in loss of conjunctival goblet cells cause dry eye symptoms, although aqueous tear production is normal: Stevens-Johnson syndrome, severe chemical injuries, or ocular pemphigoid. Certain medications, including systemic antihistamines, diuretics, antidepressants, and dermatologic drying agents, make dry eye symptoms worse.

Treatment consists of frequent use of artificial tears and, if needed, a lubricating ophthalmic ointment at bedtime. Nonpreserved artificial tears may provide better relief if patients need to use tears more frequently than every 2 hours during the day. Temporary or permanent occlusion of the lacrimal drainage apparatus may improve the efficacy of the artificial tears. Cyclosporine drops (Restasis), which improve tear production, are an effective treatment in some cases. Patients should be counseled to avoid activities that may increase the severity of dry-eye symptoms. Severe tear deficiency states are best managed by an ophthalmologist because of an increased risk of corneal ulceration.
Exposure Keratitis

Exposure keratitis causes symptoms similar to dry eyes. Exposure keratitis comes from incomplete eyelid closure during blinking, deficient blinking, or eyes coming open during sleep. Exposure may also result from Bell’s palsy, scarred or malpositioned eyelids, or thyroid exophthalmos, as the patient pictured here.

Management involves the use of ophthalmic lubricating solutions and ointments. Merely patching the eye is to be avoided because of an increased risk of corneal abrasion if the lids do not cover the eye beneath the patch. However, taping the eyelids shut at night is sometimes useful. Severe cases and those requiring surgical correction, such as a tarsorrhaphy, should be referred to an ophthalmologist.

Pinguecula/Pterygium

A pinguecula is a benign actinic change in the bulbar conjunctiva at the palpebral fissure due to sunlight exposure and drying. Scar tissue on the conjunctiva becomes red because of increased vascularity of the tissue. Pingueculae are more prevalent closer to the equator, and more common in people who spend time outdoors.
The extension of this actinic process onto the cornea is called a pterygium. A pterygium is a thin sheet of fibrovascular material that grows most commonly on the nasal side of the cornea. As a pterygium becomes apparent, it frequently becomes red and inflamed when exposed to irritants such as drying or sunlight.

Management of these lesions consists of the use of artificial tears. Patients should be counseled to use artificial tears to avoid dryness, and to wear sunglasses for protection from sun and wind. When inflammation is severe or if a pterygium is actively growing, an ophthalmologist should be consulted. When a pterygium encroaches on the visual axis, or shows active growth, it should be excised. Pterygia can sometimes recur after excision.

ANTERIOR SEGMENT DISORDERS

The anterior segment of the eye is composed of the conjunctiva, cornea, anterior chamber, and iris. Behind the iris, actually visible through the pupil, lies the lens. The ciliary body is a doughnut shaped muscle behind the base of the iris that functions in accommodation and secretes the aqueous. All these structures can cause a red eye.
Corneal Anatomy, Symptoms, and Examination

The cornea is the transparent tissue in the front of the eye through which light passes into the eye, similar to a watch crystal. The normally smooth, lustrous surface of the cornea is covered by epithelium, which has a texture similar to gelatin and is capable of regeneration in 18 hours. Beneath the epithelium is Bowman’s layer, which develops scarring whenever it is damaged. If scarring develops in the visual axis or central cornea, vision is impaired. The corneal stroma is made of collagen and comprises 95% of the corneal thickness. Finally, the internal surface of the cornea consists of Descemet’s membrane, the strongest layer of the cornea, on which grows endothelium, a single cell layer which maintains corneal clarity. The endothelium has no regenerative capacity, and damage to the endothelium from injury, inflammation, or high intraocular pressure results in corneal edema.

The cornea is the most richly innervated surface tissue in the body, and corneal nerve fibers have reflex connections with the oculomotor nerve branches that supply the circumferential muscles: the pupillary sphincter and the ciliary body. Acute corneal disorders, in addition to causing foreign-body pain, can cause a deep boring pain, photophobia, and blurred vision. Blurring is caused by pupillary miosis from contraction of the sphincter muscle; pain is caused by spasms of the ciliary body.

ACUTE CORNEAL DISORDERS: SYMPTOMS

- Eye pain
  - Foreign-body sensation
  - Deep and boring
- Photophobia
- Blurred vision
A slit-lamp biomicroscope is the standard tool for examining the anterior segment. However, in the absence of a slit lamp, the primary care physician will find useful information examining the smoothness and clarity of the corneal surface with a penlight. Note the irregular corneal light reflex and central opacity in this figure.

Fluorescein dye should be used to detect defects of the corneal epithelium, as seen in abrasions. Here a drop of sterile fluorescein dye strip is being applied to the lower fornix. With blinking, the fluorescein spreads over the cornea. The dye adheres only to defects in the corneal epithelium defect, and defects in the epithelium light up bright yellow-green under cobalt blue light.

A cobalt-blue light source can be employed with a magnifying loupe to enhance visibility. When viewed under blue light, areas of disrupted epithelium stain yellow-green against the black background of the intact epithelium, which does not stain.
A corneal abrasion causes tearing, pain, and photophobia. The patient usually has a foreign-body sensation, but it can be difficult to distinguish between that caused by a foreign body embedded in the cornea and pain due to the epithelial defect made by the foreign body. The epithelial defect produces a foreign body sensation as the lid rubs over it. If the abrasion persists, a deep, severe aching pain develops over time and is considerably worsened by exposure to light. Vision is usually blurred. It is easier for both the doctor and the patient to evaluate the eye after a drop of topical anesthetic ophthalmic solution has been applied to the eye. Again, fluorescein will stain the denuded areas of the cornea. The pupil is often miotic from ciliary body spasm.
Treatment is designed to foster rapid healing, restore patient comfort, and prevent secondary infections. Abrasions of the corneal epithelium may be managed by the primary care physician with a cycloplegic drop, such as 1% cyclopentolate, to relieve pain caused by ciliary body spasm; topical antibiotic drops (eg, fluoroquinolone, others) or ointment (erythromycin, bacitracin/polymyxin, or others). A pressure patch may be applied, although some physicians advocate no patching. One drop of topical anesthetic may be helpful, although topical anesthetics should never be prescribed for patient use because they are quite toxic to the corneal epithelium.

For patients experiencing severe pain, oral analgesics may be prescribed. The patient should be seen again in 24–48 hours, and failure to heal satisfactorily should be cause for referral to an ophthalmologist.

A pressure patch is achieved by placing two eyepads gently against the eye. The patient is then instructed to keep both eyes closed, while the pressure patch is taped firmly over the affected eye. The lower cheek should be pulled up firmly as tape is applied to keep the eye closed. The patch should remain on the eye for at least 24 hours, and follow-up by an ophthalmologist is indicated if the defect does not heal in 24 hours. A loose patch can do more harm than no patch, so care must be taken to ensure that the lids are securely closed under the patch.
Chemical Burns

A chemical burn to the eye with acid or alkali is a true ocular emergency, requiring 15 to 20 minutes of immediate irrigation with the nearest source of water available at the injury site. Further irrigation may be performed in an emergency center to normalize the pH in the eye. The nature of the chemical will dictate management thereafter.

Most acids produce the extent of their damage immediately upon contact. Of course, the more concentrated the acid, the more severe the immediate effect. Severe chemical burns denude the epithelium and blanch the vascular conjunctiva. Acid burns, after irrigation, can be managed like a severe corneal abrasion.

An alkali burn, shown here, can be more devastating to the eye because the alkaline agent dissolves the corneal tissue and continues to cause damage long after the initial chemical contact. The treating physician needs to ensure all particles of an alkaline agent are removed, or they will continue to release alkali. Following thorough irrigation, refer to an ophthalmologist emergently. Corneal melting or perforation can result from prolonged epithelial defects, and the risk of perforation persists until the epithelium is intact. Glaucoma, cataract, and chronic surface disease can occur as a later complication.
Contact Lens Overwear

Patients with contact lenses may have symptoms and complications of both conjunctivitis and corneal abrasions. The contact lens can mechanically cause an abrasion and/or introduce an infection to the cornea or conjunctiva. Simple cases of contact lens overwear are managed similarly to corneal abrasion, with care taken to watch for infection. Occasionally, contact lens-induced corneal abrasions, especially those associated with soft lenses, rapidly progress to a severe bacterial corneal ulcer (see figure on left). A more common complication of soft contact lens wear is giant papillary conjunctivitis (GPC; see figure on right). GPC is characterized by hypertrophic papillae on the upper tarsal plate.

Patients with contact-lens related symptoms should be seen again the next day and referred if not improved. Contact lens wear may be resumed only after the corneal epithelium has healed, and patients should be counseled not to wear the contact lens if any symptoms persist.

Infectious Keratitis

The cornea is subject to two types of injury: Mechanical trauma, such as abrasions and foreign bodies; and chemical trauma, such as acid or alkali burns. Both types of trauma can predispose to corneal infection by disrupting the protective barrier of the corneal epithelium. Because infections can result in permanent scarring and decreased vision, early detection and aggressive therapy are important.
**Bacterial Keratitis**

Bacterial infection of the cornea presents as a red, painful eye with purulent discharge, usually associated with decreased vision. Examination by penlight may reveal a discrete white or gray corneal opacity. Emergency referral to an ophthalmologist for diagnosis and treatment is required.

**Viral Keratitis**

Primary herpes simplex ocular infection usually presents as a unilateral foreign-body sensation with watery discharge. There may be skin vesicles on the lids or enlarged preauricular lymph nodes. The herpes simplex virus resides in the trigeminal ganglia, and recurrent outbreaks of herpetic lesions result from periodic reactivation of the virus.

Corneal involvement by herpes simplex virus is usually unilateral and typically presents with a red, tearing eye with foreign-body sensation. Epithelial dendrites, characteristic of this condition, are small arborizing epithelial lesions in the shape of a twig or branch. When a corneal dendrite is detected by staining with fluorescein, the patient should be immediately referred to an ophthalmologist.
Under no condition should topical anesthetic solutions be given to the patient or prescribed for pain relief. The toxic effects of repeated administration of topical anesthetics on the corneal epithelium can cause permanent scarring and loss of vision. Anesthetic drops should not be prescribed for patients because of this risk.

Additionally, topical steroids should be prescribed only by an ophthalmologist because of their four potentially serious ocular side effects:

- Topical corticosteroid drops can potentiate a latent herpes simplex infection of the cornea. Steroids can also facilitate penetration of the herpes infection to the deeper layers of the cornea, resulting in permanent corneal scarring or perforation.
- Local use of steroids can elevate intraocular pressure in susceptible individuals, possibly effecting steroid-induced glaucoma.
- Topical corticosteroid drops over time can cause cataracts to progress faster than usual.
- The misuse of steroids is capable of potentiating the development of fungal ulcers of the cornea.

**TOPICAL STEROIDS: SIDE EFFECTS**

- Facilitate corneal penetration of herpes virus
- Elevate IOP (steroid-induced glaucoma)
- Cataract formation and progression
- Potentiate fungal corneal ulcers
Hyphema

Blunt trauma to the eye can cause injury to the iris, anterior chamber angle, or ciliary body and result in a hyphema. Blood in the anterior chamber can layer out if the patient has been relatively immobile, or if the patient is active, it can be stirred up and obliterate a clear view of the iris. Vision is usually decreased, and pain and redness may be present. The patient shown here has maintained an upright position, and the red blood cells have settled in the eye. A hyphema is an ocular emergency, and the patient should be referred immediately to an ophthalmologist.

Inflammatory Conditions

Inflammation in the eye includes diseases such as episcleritis, scleritis, and uveitis (or iritis). These diseases typically have an autoimmune component and occasionally may be associated with systemic disease. Treatment is usually with topical or oral corticosteroids or other anti-inflammatory drugs.

**INFLAMMATORY CONDITIONS CAUSING A RED EYE:**
- Episcleritis
- Scleritis
- Anterior uveitis (iritis)
Episcleritis and scleritis are inflammatory conditions that present with eye pain and redness. Both conditions have several variants and can present with sectoral, diffuse, or nodular inflammation. Episcleritis is an inflammation of the superficial episcleral vessels and usually causes relatively mild ocular discomfort. Although episcleritis can be associated with systemic autoimmune disorders, it is most commonly idiopathic. Scleritis is an inflammation of the sclera and deeper episcleral vessels and is often associated with more severe pain. An underlying autoimmune disorder can be found in up to 50% of patients with scleritis, most commonly rheumatoid arthritis. Although episcleritis often can be managed with topical steroids or nonsteroidal drops, patients with scleritis often require additional systemic anti-inflammatory treatment with oral nonsteroidal anti-inflammatory drugs, oral steroids, or in some cases, other immunosuppressive agents.

A patient with iritis may present with circumlimbal redness, pain, photophobia, and decreased vision. The pupil is usually smaller than the contralateral eye due to ciliary body spasm. Iritis frequently accompanies other inflammatory conditions, including infections, arthritis, and sarcoidosis, urethritis, and bowel disorders. Iritis may also occur as a result of blunt trauma to the eyes. In such cases, signs and symptoms usually begin one to several days following trauma.
Uveitis can be very painful, and the pain does not always localize to the eye. Frequently the pain is a boring deep pain like an "ice cream" headache that feels like it is coming from behind the eye. Untreated or improperly treated iritis can be complicated by the development of glaucoma and cataracts. Early recognition of this clinical picture and prompt referral are essential.

Acute uveitis is characterized by white blood cells in the anterior chamber. These cells are generally best seen with slit-lamp biomicroscopy of the anterior chamber. In severe cases, the cells may collect in the interior portion of the anterior chamber and form a hypopyon. In cases of chronic uveitis, white blood cells may collect on the corneal endothelial surface and form keratic precipitates sometimes called “mutton fat.”

Acute Angle-Closure Glaucoma

In the normal eye, aqueous humor flows through the pupil into the anterior chamber where it is drained primarily through the trabecular meshwork to a canal leading to the venous system (left). Acute elevations in intraocular pressure can occur when the peripheral iris occludes the trabecular meshwork in the angle and suddenly blocks the outflow of aqueous humor from the anterior chamber (right). Such an attack may occur following dilation of the pupil in dim lighting or an instillation of dilating eye drops. Even emotional stress or systemic medications that dilate the pupil can sometimes trigger an attack in susceptible individuals.
The pain of angle-closure glaucoma is among the worst the body can experience. Patients experiencing an acute attack of angle-closure glaucoma complain of severe ocular pain, frontal headache, blurred vision, and the appearance of halos around lights. Nausea and vomiting are often present. Generally, the symptoms are displayed in one eye only, although both eyes are usually predisposed to this condition.

The easiest way to rule out angle-closure glaucoma is to check the intraocular pressure. Although most primary care physicians are not familiar with these techniques, the availability of a tonometer such a Schiøtz or TonoPen in patient-care settings where angle-closure glaucoma might be encountered would make diagnosis easier. In the absence of these tools, a penlight examination of the affected eye would reveal a pupil fixed in mid-dilation and slightly larger than the contralateral pupil; a responsive pupil during acute angle closure would be unusual. Often the cornea appears hazy or “steamy” due to edema.

An acute episode of angle-closure glaucoma is an ocular emergency and requires immediate intervention. Beware of the trap of confusing this uncommon ophthalmic entity with a cerebral aneurysm (which is accompanied by headaches and a fixed, dilated pupil) or with abdominal pathology (symptoms of which include nausea, vomiting, and usually abdominal pain), because evaluation of these entities only delays the needed ophthalmic treatment.
If an ophthalmologist cannot attend a patient with acute angle-closure glaucoma within the hour, the primary care physician should initiate treatment. This should include administering topical 2% pilocarpine drops in two doses, 15 minutes apart. Topical timolol maleate 0.5%, a beta blocker, and topical apraclonidine 0.5%, an alpha-adrenergic agonist, may also be administered. Systemically, acetazolamide, 500 mg orally or parenterally, should be given. A 20% solution of IV mannitol is sometimes necessary. The longer the intraocular pressure remains high, the greater the risk of permanent visual loss. Improved comfort suggests that the pressure is becoming lower, as do the return of pupillary movement and the resolution of stromal edema.

**SUMMARY**

To summarize, many conditions may present with a red eye or red lid. Hordeolum, chalazion, blepharitis, conjunctivitis, subconjunctival hemorrhage, dry eyes, and corneal abrasions can usually be diagnosed easily and treated by the primary care physician.

**ACUTE GLAUCOMA: INITIAL TREATMENT**

- Pilocarpine 2% drops q 15 min x 2
- Timolol maleate 0.5%, 1 drop
- Apraclonidine 0.5%, 1 drop
- Acetazolamide 500 mg po or IV
- IV mannitol 20% 300–500 cc

**COMMON RED EYE DISORDERS: TREATMENT INDICATED**

- Hordeolum
- Chalazion
- Blepharitis
- Conjunctivitis
- Subconjunctival hemorrhage
- Dry eyes
- Corneal abrasions (most)
However, when the physician notes decreased vision, ocular pain, photophobia, circumlimbal redness, corneal edema, corneal opacities or dendrites, or an abnormal pupil, the patient should be referred to an ophthalmologist.

Orbital cellulitis, episcleritis, scleritis, chemical injury, corneal infection, hyphema, iritis, and acute angle-closure glaucoma are urgent conditions that threaten vision and require immediate referral to an ophthalmologist.

In conclusion, the successful management of the red eye depends on the clinical expertise of the primary care physician and close cooperation and communication between the primary care physician and the ophthalmologist. Early diagnosis and treatment can reduce patient morbidity, and reduce the chance of permanent vision loss.
APPENDIX 1
Common Red Eye Disorders: Diagnosis and Management

I. Ocular Adnexa

A. Hordeolum/chalazion: Inflamed glands of lid due to occluded orifices of Meibomian glands (often complicates blepharitis)
   1. Symptoms/signs: may present as localized or diffuse cellulitis of lid, associated with tenderness
   2. Treatment
      a. Treat blepharitis if present
      b. Warm compresses for 10 mins tid when acute or subacute; continue until resolved (may take several weeks)
      c. Refer to ophthalmologist if chalazion fails to resolve and becomes chronic, ie, nontender, localized

B. Blepharitis: A chronic lid margin inflammation
   1. Associated with:
      a. Staphylococcal infection
      b. Seborrhea
      c. Dry eyes
   2. Symptoms/signs
      a. Burning
      b. Foreign-body sensation
      c. Thick, red lid margins with crusting
      d. Lids often sticking in AM
      e. May be asymptomatic
   3. Treatment
      a. Warm compresses to loosen crusting
      b. Proper lid hygiene: scrub lids thoroughly with warm washcloth, plus nonirritating shampoo in AM and hs
      c. Topical ophthalmic antibiotic ointment hs x 2–3 weeks (eg, erythromycin) or antibiotic/steroid ointment
      d. Oral antibiotics (tetracycline or erythromycin) in refractory cases only

C. Cellulitis of extraocular structures
   1. Anterior (periorbital or preseptal) cellulitis
      a. Symptoms/signs
         i. Swollen, red lids and skin
         ii. Lids may be tender
         iii. Vision, pupils, ocular motility are normal
      b. Treatment
         i. Warm compresses
         ii. Systemic antibiotics
   2. Posterior (orbital) cellulitis
      a. Symptoms/signs
         i. Swollen, red lids and conjunctiva
         ii. Periorbital area relatively uninflamed
iii. Impaired ocular motility with pain on eye movement
iv. Proptosis
v. If optic nerve involvement: decreased vision, afferent pupillary defect, optic disc edema

b. Management
i. Hospitalization
ii. Stat ophthalmology consultation
iii. Blood culture
iv. Orbital CT scan
v. ENT consultation if sinus disease present
vi. IV antibiotics stat (Staphylococcus aureus, Streptococcus species, Haemophilus influenzae most common)
vii. Rule out fungal infection in immunosuppressed patient (may require surgical debridement)
viii. Surgery if no rapid response to IV antibiotics or if subperiosteal abscess present
ix. Watch for complications: cavernous sinus thrombosis, meningitis

II. Lacrimal System

A. Nasolacrimal duct obstruction
1. Symptoms/signs
   a. Persistent tearing and discharge, often associated with a red eye
   b. ± Dacryocystitis (infected tear sac)
2. Treatment: congenital obstructionMassage lacrimal sac daily
   a. Systemic antibiotics if dacryocystitis
   b. Refer to ophthalmologist if no resolution in 6–8 months
3. Acquired obstruction
   a. Systemic antibiotics if dacryocystitis
   b. Chronic/recurrent: Refer to ophthalmologist

III. Ocular Surface

A. Conjunctivitis (adult)
1. Symptoms/signs
   a. Pattern of redness: palpebral or diffuse
   b. Discharge: characteristic of cause
      i. Purulent: bacterial
      ii. Watery, serous: viral
      iii. Watery, with white, stringy mucus: allergic
2. Bacterial conjunctivitis
   a. Most common: S. aureus, Streptococcus species, H. influenzae
   b. Treatment
      i. Warm compresses
      ii. Topical antibiotics qid x 7 days
      iii. Refer to ophthalmologist if not improved in 3 days
   c. Copious purulent discharge
      i. Stat Gram’s stain, culture (rule out N. gonorrhoeae)
      ii. Refer to ophthalmologist
3. Viral conjunctivitis
   a. Contagious (adenovirus)
b. No effective therapy except time (1½–6 weeks)
c. Refer to ophthalmologist if pain, photophobia, decreased vision or if condition persists 2+ weeks

4. Allergic conjunctivitis
   a. Itching, burning eyes
   b. ± Lid/conjunctival edema
   c. ± White, stringy mucus
d. Treatment: symptomatic
   i. Topical antihistamines or artificial tears
   ii. Refer if refractory to treatment

B. **Conjunctivitis (neonatal):** culture and smear to differentiate types
1. Bacterial
   a. "Beefy redness" indicates *N. gonorrhoeae*; refer urgently
2. Chlamydial
   a. Mild unilateral or bilateral mucopurulence
   b. Moderate lid edema and infection
c. Erythromycin ointment: qid x 4 weeks
d. Erythromycin po x 2–3 weeks 40–50 mg/kg/day ÷ 4

C. **Subconjunctival hemorrhage**
1. Usually spontaneous, without known cause
2. Possible association with anticoagulants, aspirin, or high-dose vitamin E
3. Patient often presents with bright red eye, normal vision, no pain
4. Examine carefully to rule out traumatic cause (perforating injury)
5. No treatment except time (2–3 weeks) and reassurance

D. **Dry eyes**
1. Tear deficiency (keratoconjunctivitis sicca)
   a. Burning, “gritty” sensation (symptoms exceed signs)
b. Common with aging
c. Associated conditions
   i. Rheumatoid arthritis; Stevens-Johnson syndrome; ocular pemphigoid; systemic medications (diuretics, antihistamines, antidepressants, dermatologic drying agents)
d. Treatment
   i. Artificial tears instilled frequently or cyclosporine drops
   ii. Lubricating ophthalmic ointment hs
   iii. ± Punctal occlusion
e. If severe or unresponsive to simple measures, refer to an ophthalmologist
2. Exposure keratitis
   a. Causes: Bell’s palsy, scarred or malpositioned lids, thyroid exophthalmos
   b. Treatment (if inflamed)
   i. Artificial tears, lubricating ointment
   ii. Tape lids shut hs prn; do not patch
   iii. Refer if severe

E. **Pinguecula/pterygium:** A benign actinic change caused by exposure to sun, wind
1. Arises from bulbar conjunctiva at palpebral fissure (nasal and/or temporal)
2. Pinguecula: confined to conjunctival tissue
3. Pterygium: extension onto the cornea-
4. Treatment
   a. Frequent use of artificial tears
   b. Topical ophthalmic solutions with vasoconstrictors qid prn to alleviate redness
c. Refer to ophthalmologist if actively growing pterygium is present or if inflammation is severe
IV. Anterior Segment

A. Corneal abrasion
   1. Symptoms/signs: redness, tearing, photophobia, pain; foreign-body sensation initially; blurred vision
   2. Treatment
      a. Relieve pain with cycloplegic drops (1% cyclopentolate, 5% homatropine); oral analgesics with codeine if severe pain
      b. Prevent infection with topical antibiotic
      c. Promote rapid healing with pressure patch (2 eye pads) for at least 24 hours
   3. Refer to ophthalmologist in 24–48 hours if not healed

B. Chemical injury
   1. Acid
      a. Causes immediate damage
      b. Stat irrigation
      c. Refer to ophthalmologist
   2. Alkali
      a. Causes immediate and delayed damage; potential for serous ocular damage
      b. Stat irrigating
      c. Stat referral to ophthalmologist

C. Contact lens overwear
   1. Treat the same as corneal abrasion, but avoid patching if soft contact lens wearer
   2. Watch for development of corneal ulcer or GPC

D. Keratitis
   1. Viral: herpes simplex type I most common
      a. Symptoms/signs
         i. Red eye with watery discharge and foreign-body sensation
         ii. Dendrite or branching figure, characteristic epithelial lesion of cornea; best seen with fluorescein stain
      b. Refer to ophthalmologist stat
   2. Bacterial
      a. Symptoms/signs
         i. Red, painful eye with purulent discharge and decreased vision
         ii. Discrete corneal opacity seen with penlight
      b. Refer to ophthalmologist stat

E. Hyphema: Blood in the anterior chamber
   1. Usually follows blunt trauma
   2. Symptoms/signs: decreased vision, pain, redness, blood in the anterior chamber
   3. Refer to ophthalmologist stat

F. Episcleritis/scleritis
   1. Localized redness and tenderness but diffuse variations
   2. Most cases are idiopathic
   3. ± Associated conditions: autoimmune disorders, eg, rheumatoid arthritis
   4. Scleritis can lead to vision-threatening complications
   5. Always refer to an ophthalmologist

G. Iritis: Inflammation of the anterior chamber
   1. Symptoms/signs: circumlimbal redness, eye pain, ± boring headache, photophobia, decreased vision, and small pupil; hypopyon, keratic precipitates
   2. ±Associated conditions: infections, arthritis, sarcoidosis, and urethritis, inflammatory bowel disorders.
   3. Onset following blunt trauma to eye, usually delayed 1–3 days
4. Complications: glaucoma and cataract
5. Recognize and refer to an ophthalmologist promptly

H. **Acute angle-closure glaucoma:** Sudden block of aqueous outflow
1. Characteristically seen in susceptible individuals who experience acute rise in IOP when pupil dilates
2. Predicating factors: dim light, some pharmacologic agents (topical and systemic), emotional stress
3. Symptoms
   a. Severe ocular pain
   b. Frontal headache
   c. Blurred vision
   d. Perception of halos around lights
   e. ±Nausea and vomiting
4. Signs
   a. Redness
   b. Mid-dilated, nonreactive pupil
   c. Cloudy cornea
   d. Affected eye appreciably hard on palpation
   e. Usually one eye is involved
5. Can be confused with other conditions, eg, cerebral aneurysm (headache, fixed dilated pupil), appendicitis (nausea, vomiting)
6. Refer to ophthalmologist stat
7. If treatment by ophthalmologist is to be delayed by 1 hour or more, primary care physician should begin treatment:
   a. 2% pilocarpine eye drops q 15 mins x 2
   b. Timolol maleate 0.5%, 1 drop
   c. Apraclonidine 0.5%, 1 drop
   d. Acetazolamide 500 mg po or IV
   e. ±20% solution IV mannitol

V. Vision-Threatening Red Eye Disorders

A. **Symptoms/signs**
   1. Decreased vision
   2. Ocular pain
   3. Photophobia
   4. Circumlimbal redness
   5. Corneal edema
   6. Corneal ulcers, dendrites
   7. Abnormal pupil
   8. Proptosis
   9. Elevated intraocular pressure

B. **Conditions:** Recognize and refer
   1. Orbital cellulitis
   2. Episcleritis, scleritis
   3. Chemical injury
   4. Corneal infection
   5. Hyphema
   6. Iritis
   7. Acute glaucoma
# APPENDIX 2
## The Red Eye: Differential Diagnosis

<table>
<thead>
<tr>
<th></th>
<th>Conjunctivitis</th>
<th>Iritis</th>
<th>Keratitis (Corneal Inflammation or Foreign Body)</th>
<th>Acute Angle-Closure Glaucoma</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vision</strong></td>
<td>Normal or intermittent&lt;br&gt;Blurring that clears on blinking</td>
<td>Slightly blurred</td>
<td>Slightly blurred</td>
<td>Marked blurring</td>
</tr>
<tr>
<td><strong>Discharge</strong></td>
<td>Usually significant, with crusting of lashes</td>
<td>None</td>
<td>None to mild</td>
<td>None</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td>None or minor and superficial</td>
<td>Moderately severe: aching and photophobia</td>
<td>Sharp, severe foreign-body sensation</td>
<td>Very severe, frequently nausea and vomiting</td>
</tr>
<tr>
<td><strong>Pupil size</strong></td>
<td>Normal</td>
<td>Constricted</td>
<td>Normal or constricted</td>
<td>Fixed, dilated</td>
</tr>
<tr>
<td><strong>Conjunctival injection</strong></td>
<td>Diffuse</td>
<td>Circumcorneal</td>
<td>Circumcorneal</td>
<td>Diffuse, with prominent circumcorneal injection</td>
</tr>
<tr>
<td><strong>Pupillary response to light</strong></td>
<td>Normal</td>
<td>Minimal further constriction</td>
<td>Normal</td>
<td>Usually no reaction of mid-dilated pupil</td>
</tr>
<tr>
<td><strong>Intraocular pressure</strong></td>
<td>Normal</td>
<td>Normal to low</td>
<td>Normal</td>
<td>Markedly elevated to touch</td>
</tr>
<tr>
<td><strong>Appearance of Cornea</strong></td>
<td>Clear</td>
<td>Clear or slightly hazy</td>
<td>Opacification present; altered light reflex; positive fluorescein staining</td>
<td>Hazy; altered light reflex</td>
</tr>
<tr>
<td><strong>Anterior chamber depth</strong></td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Shallow</td>
</tr>
</tbody>
</table>

*SPECIAL NOTE ON ACUTE ANGLE-CLOSURE GLAUCOMA:* It is highly desirable for an ophthalmologist to examine the patient during an acute attack to confirm the diagnosis.
APPENDIX 3

Resources


Basic and Clinical Science Course, Section 8: External Disease and Cornea. San Francisco: American Academy of Ophthalmology; (updated annually).


