Chapter 2

OPHTHALMIC CONDITIONS

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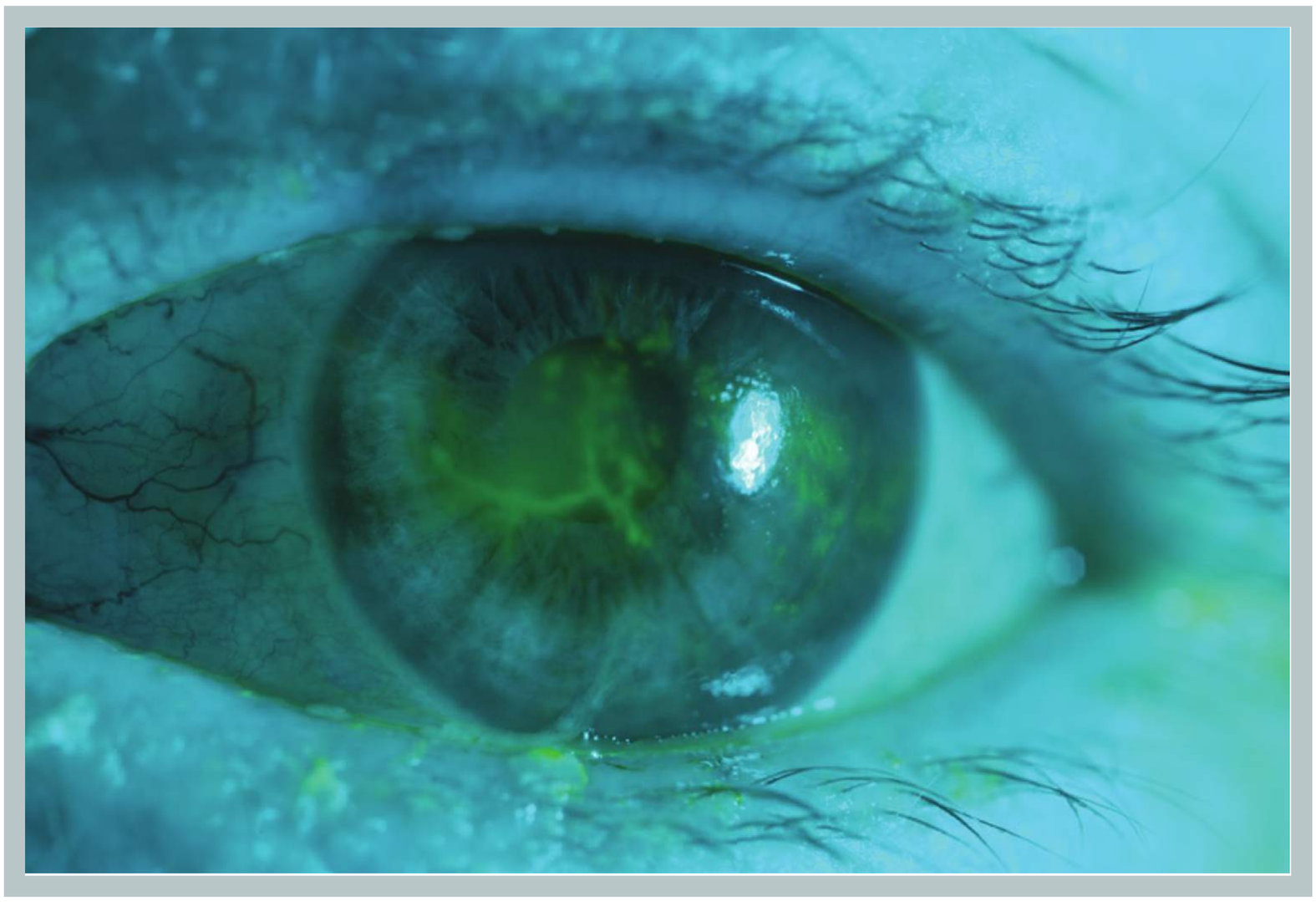

Herpes Simplex Keratitis. Branched dendritic lesion seen on the cornea in a patient with HSV. (Photo contributor: Lawrence B. Stack, MD.)

The authors acknowledge the special contribution of Marc E. Levsky, MD, and Paul DeFlorio, MD, for portions of this chapter written for the previous editions of this book.

NEONATAL CONJUNCTIVITIS (OPHTHALMIA NEONATORUM)

Clinical Summary

Neonatal conjunctivitis is acquired either during birth with passage through the mother's cervix and vagina, or from cross-infection in the neonatal period. Presenting symptoms for Neisseria gonorrhoeae infection include a hyperacute bilateral conjunctivitis with copious purulent discharge, lid swelling, chemosis, and preauricular adenopathy.

More common etiologies include Chlamydia trachomatis, viruses (herpes simplex), and bacteria (Staphylococcus aureus, Streptococcal pneumoniae, Haemophilus species). For chlamydial conjunctivitis, the clinical features range from mild swelling with a watery discharge to marked lid swelling with a red and thickened conjunctiva with a blood-stained discharge. Fluorescein staining of herpes simplex conjunctivitis demonstrates epithelial dendrites.

Management and Disposition

With any form of neonatal conjunctivitis, Gram stain and culture are indicated. Scrapings of the palpebral conjunctiva are more likely to be rewarding than examination of the discharge itself. Begin treatment in the emergency department (ED) and admit newborns with suspected gonococcal conjunctivitis. Evaluate concurrently for C trachomatis, since coinfection is common.

Treatment for chlamydial conjunctivitis is based upon a positive diagnostic test. While culture is the gold standard, nucleic acid amplification tests, despite lacking FDA approval, are reported to perform similarly. Untreated disease can result in corneal and conjunctival scarring. Bacterial neonatal conjunctivitis that is neither gonococcal nor chlamydial may be treated with erythromycin antibiotic ointment and should be reevaluated in 24 hours.

Herpes simplex conjunctivitis is treated with intravenous (IV) acyclovir and topical trifluridine. Despite the appearance of a localized herpes infection, there is high risk for central nervous system (CNS) or disseminated infection.

Evaluation of the newborn's parents should be undertaken in neonatal conjunctivitis due to Gonococcus, Chlamydia, or herpes simplex virus (HSV).

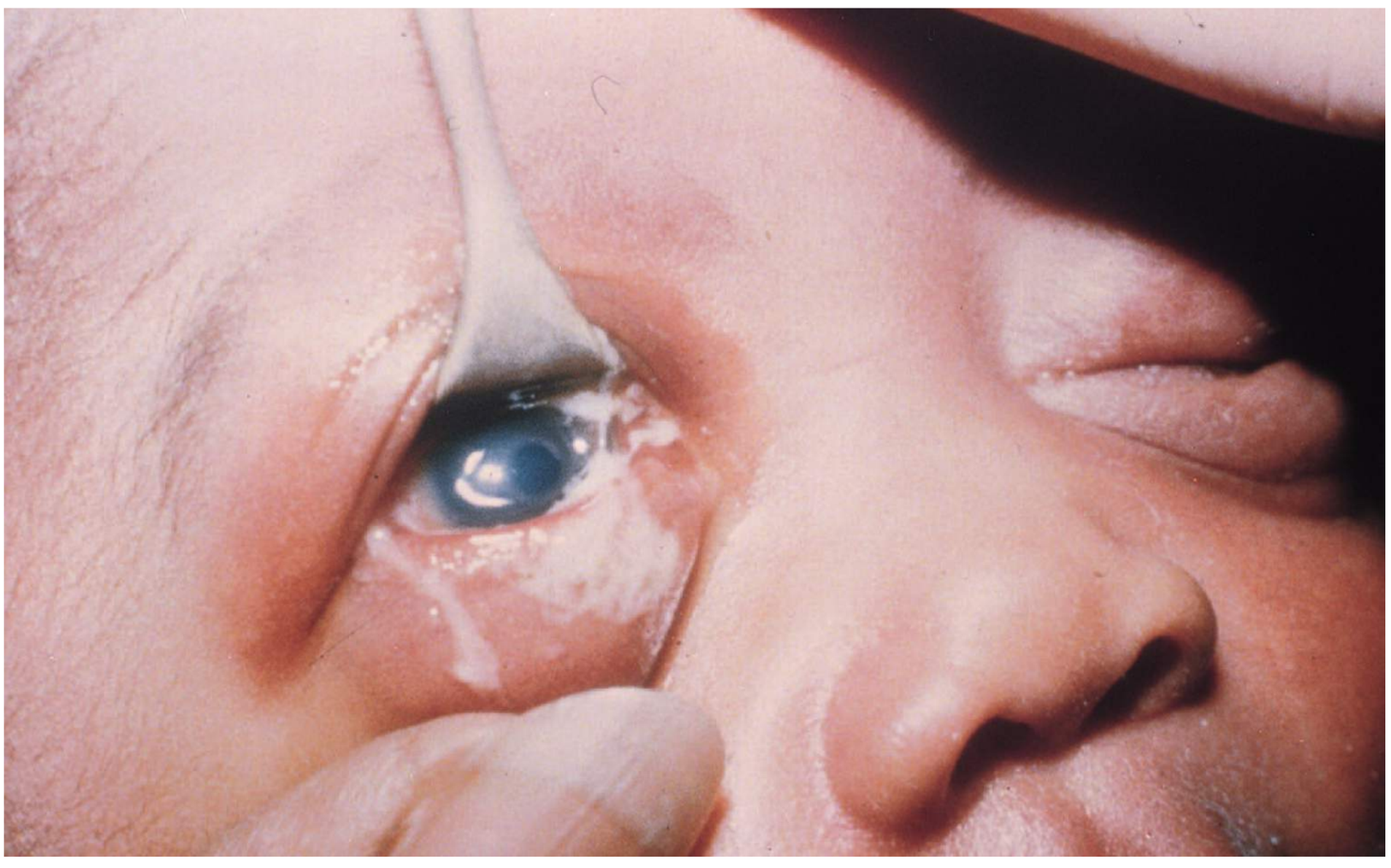

FIGURE 2.1 ■ **Neonatal Conjunctivitis (Ophthalmia Neonatorum).** Copious purulent drainage in a newborn with neonatal gonococcal conjunctivitis. (Reprinted with permission of the American Academy of Ophthalmology. Eye Trauma and Emergencies: A Slide-Script Program. San Francisco, 1985.)

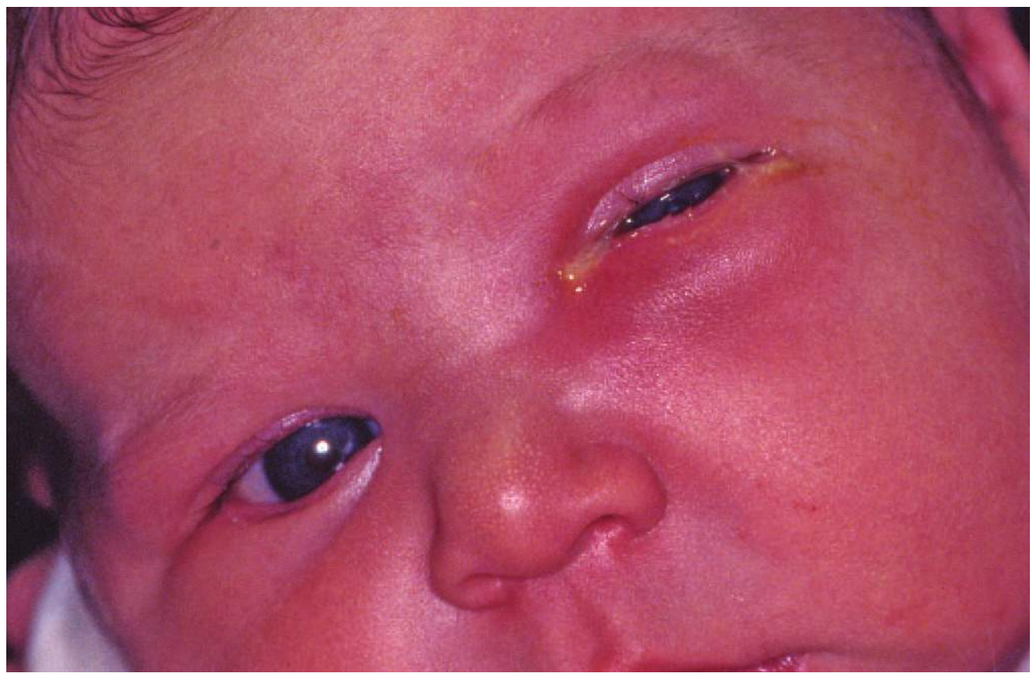

FIGURE 2.2 ■ **Neonatal Conjunctivitis.** Thick purulent drainage in a newborn diagnosed with neonatal gonococcal conjunctivitis. (Photo contributor: David Effron, MD.)

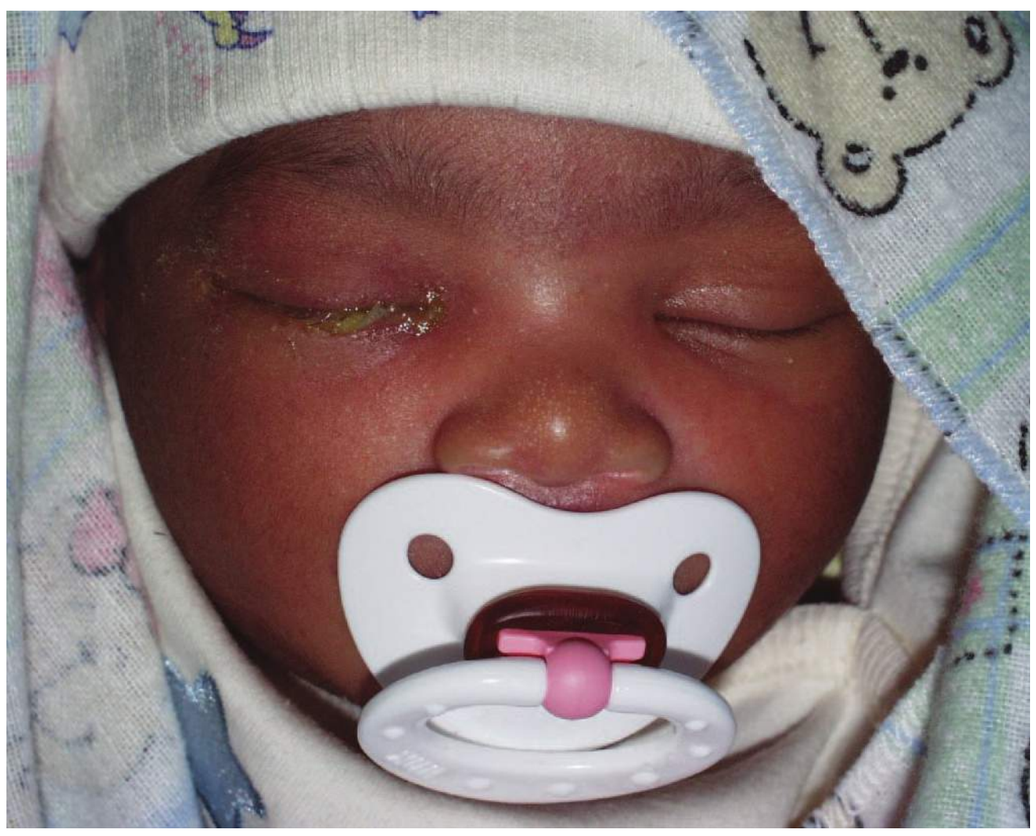

FIGURE 2.3 ■ **Neonatal Conjunctivitis.** A purulent discharge is seen in this newborn. Management includes excluding Neisseria and Chlamydia. (Photo contributor: Kevin J. Knoop, MD, MS.)

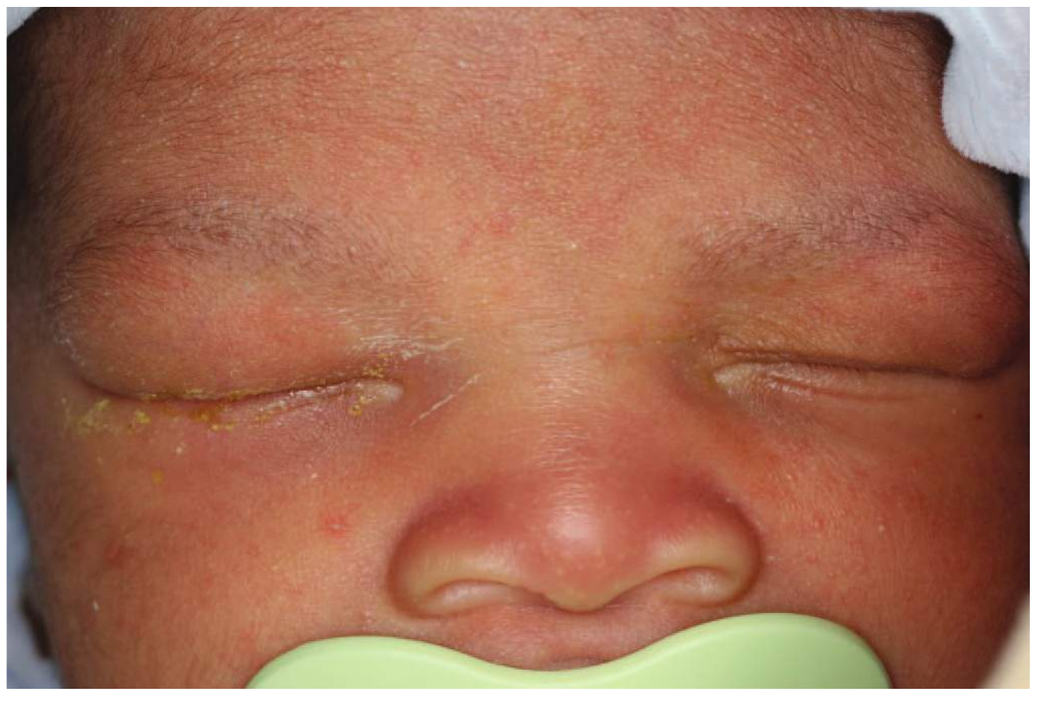

FIGURE 2.4 ■ **Neonatal Conjunctivitis.** A scant crusty discharge is seen in this newborn who was diagnosed in follow-up with nasolacrimal duct obstruction. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. The "rule of fives" may help predict the most likely bacterial cause.

0-5 days	Gonococcus
5 days-5 weeks	Chlamydia
5 weeks-5 years	Staphylococcus, Streptococcus, Haemophilus

2. Blindness can result from gonococcal eye infection in the neonate because the organism can invade the cornea. It is one of the few emergency conjunctival infections.
3. Nasolacrimal duct obstruction is common (up to 20%) in newborns and may present with findings suggestive of conjunctivitis. It is a diagnosis of exclusion in the neonate.
4. Advise parents that infants treated with macrolides are at risk for developing hypertrophic pyloric stenosis.

TABLE 2.1 ■ SUMMARY OF NEONATAL CONJUNCTIVITIS

Etiologic Agent	Time of onset	Clinical Features	Treatment
Chemical	Day 1	Common with silver nitrate; erythromycin more commonly used now for this reason	Self-limited
N gonorrhoeae	Day 2-7	May cause hyperacute disease with profuse discharge	Ceftriaxone IV. Caution in hyperbilirubinemia. Topicals alone inadequate
C trachomatis	Day 3-14	Clinical severity varies. Common cause of blindness worldwide	Erythromycin PO Azithromycin (alternate)
HSV 1,2	Day 2-16	Should be suspected if child has any vesicular lesions on body	Vidarabine or trifluridine topically; consider systemic acyclovir
Other bacterial organisms	Day 2 and up	Empiric treatment based on Gram stain	Erythromycin ointment for gram positive; gentamicin or tobramycin ointment for gram negative

BACTERIAL CONJUNCTIVITIS

Clinical Summary

Bacterial conjunctivitis is characterized by the acute onset of conjunctival injection and a thick yellow, white, or green mucopurulent drainage. Lid edema, erythema, and chemosis may also be seen. S aureus is the most common causative bacteria. Streptococcus pneumoniae and Haemophilus influenzae occur more frequently in children.

Hyperacute bacterial conjunctivitis, the most severe form of acute purulent conjunctivitis, is associated with N gonorrhoeae. Symptoms are hyperacute in onset, and findings include a purulent, thick, copious discharge, eyelid swelling and tenderness, marked conjunctival hyperemia, chemosis, and preauricular adenopathy. The condition is serious and threatens sight because Neisseria species are capable of invading an intact corneal epithelium. Corneal findings include epithelial defects, marginal infiltrates, and an ulcerative keratitis that can progress to perforation.

Management and Disposition

Encourage frequent hand washing and warm moist compresses. Empiric antibiotic choices include polymyxin/trimethoprim drops and erythromycin ophthalmic ointment. Other options include bacitracin, sulfacetamide, or polymyxin-bacitracin ointments or fluoroquinolone or azithromycin drops. Improvement should be noted in 1 to 2 days. Patients who do not improve should be referred to an ophthalmologist.

Fluoroquinolones should be prescribed for contact lens wearers (and keratitis ruled out) due to concern for Pseudomonas infection. Hyperacute bacterial conjunctivitis requires immediate ophthalmologic consultation and hospitalization.

FIGURE 2.5 ■ **Bacterial Conjunctivitis.** Mucopurulent discharge, conjunctival injection, and lid swelling in a 10-year-old with H influenzae conjunctivitis. (Photo contributor: Frank Birinyi, MD.)

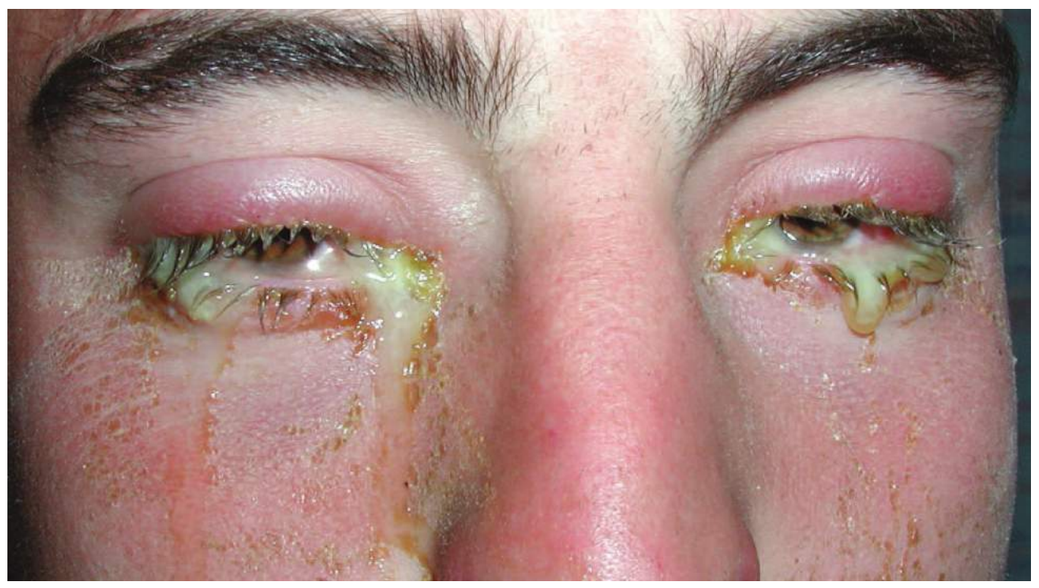

FIGURE 2.6 ■ **Bacterial Conjunctivitis.** Mucopurulent discharge and conjunctival injection in an adult with conjunctivitis. (Photo contributor: Lawrence E. Heiskell, MD.)

Pearls

1. Conjunctivitis is a common presentation of the red eye, and should be considered after more serious causes, such as acute angle-closure glaucoma (ACG), iritis, and infectious keratitis, have been ruled out.
2. Red flags include a significantly decreased visual acuity, ciliary flush, and corneal opacity.
3. Worsening symptoms during topical treatment with Neosporin or a sulfonamide suggest a contact allergic reaction.
4. N gonorrhoeae conjunctivitis must be considered in the sexually active adult with a prominent, thick, copious discharge. Urethritis is usually present.
5. Fluoroquinolones should be prescribed for contact lens wearers because of concern for Pseudomonas infection in these patients.

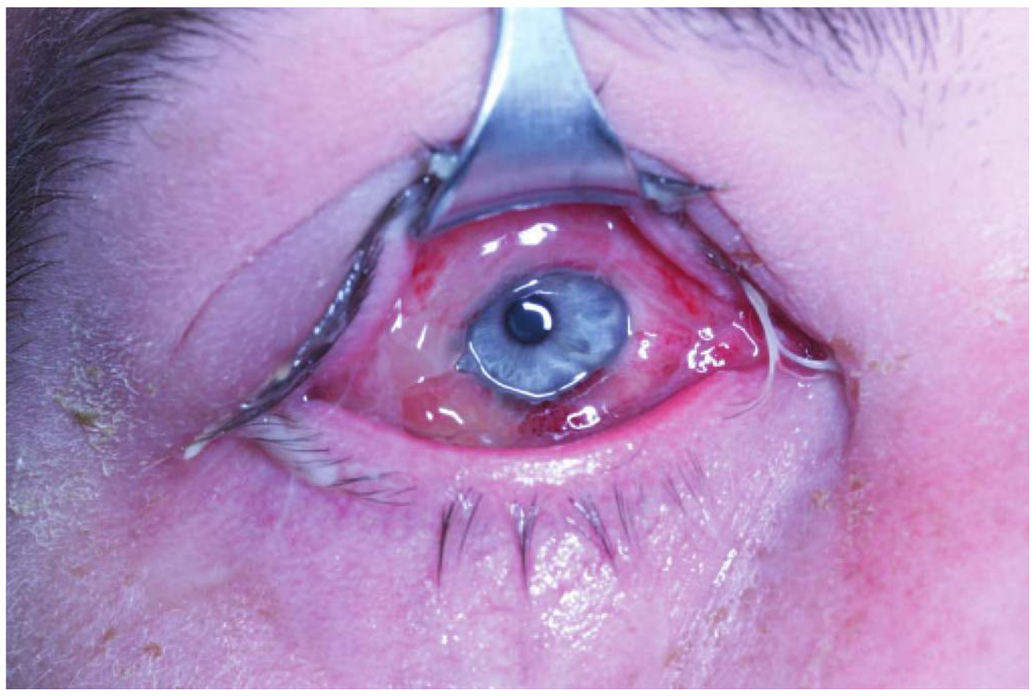

FIGURE 2.7 ■ **Gonococcal Conjunctivitis.** Right eye chemosis, purulent discharge, and pain in young adult male with urethritis. (Photo contributor: Catherine Burger, MD.)

Clinical Summary

Viral conjunctivitis is a common presentation of the red eye. Findings are mild and include a thin watery discharge, crusting in the morning, burning or irritation, conjunctival injection (typically diffuse), and lid edema. The tarsal conjunctiva may appear bumpy secondary to hyperplastic lymphoid tissue (follicles). Preauricular adenopathy may be present. The visual acuity is normal. The infection usually begins in one eye, but both eyes usually become involved due to autoinoculation. There are few to no systemic complaints. Adenovirus is the most common virus. A point of care test now available may aid clinicians to avoid empiric antibiotic therapy.

Epidemic keratoconjunctivitis (EKC) is a severe and highly contagious adenovirus infection that also involves the cornea. Additional features may include foreign body sensation, photophobia, and pseudomembranes overlying the palpebral conjunctiva. By the eighth day, a painful punctate keratitis that stains with fluorescein may develop; by the end of the second week, these are replaced by white macular subepithelial infiltrates located in the central cornea (that no longer stain). These may cause a decrease in visual acuity, but eventually resolve spontaneously.

Pharyngoconjunctival fever, usually caused by adenovirus type 3, is highly contagious and should be considered if there is associated upper respiratory tract infection and fever.

Management and Disposition

Viral conjunctivitis is self-limited and usually mild. Warm or cool compresses may be helpful. Careful hand washing by patient and staff is important. Over-the-counter topical antihistamines (Naphcon A) may provide symptomatic relief. The eye irritation and discharge should improve after 5 to 7 days, but may take 2 to 3 weeks for complete resolution of all symptoms. Antivirals and antibiotics are ineffective. Patients with keratitis should follow up with an ophthalmologist.

Pearls

1. A nonspecific adenovirus conjunctivitis will improve after 5 to 7 days and resolve in 10 to 14 days, but a virulent adenovirus causing EKC will peak in 5 to 7 days and may last 3 to 4 weeks.
2. Consider and rule out serious causes of red eye (acute ACG, iritis).
3. Frequent hand washing and the use of separate linens are advised for patients and family members.
4. Fastidious hand and equipment hygiene is necessary to prevent nosocomial transmission, as adenovirus can be recovered for extended periods of time from these surfaces.

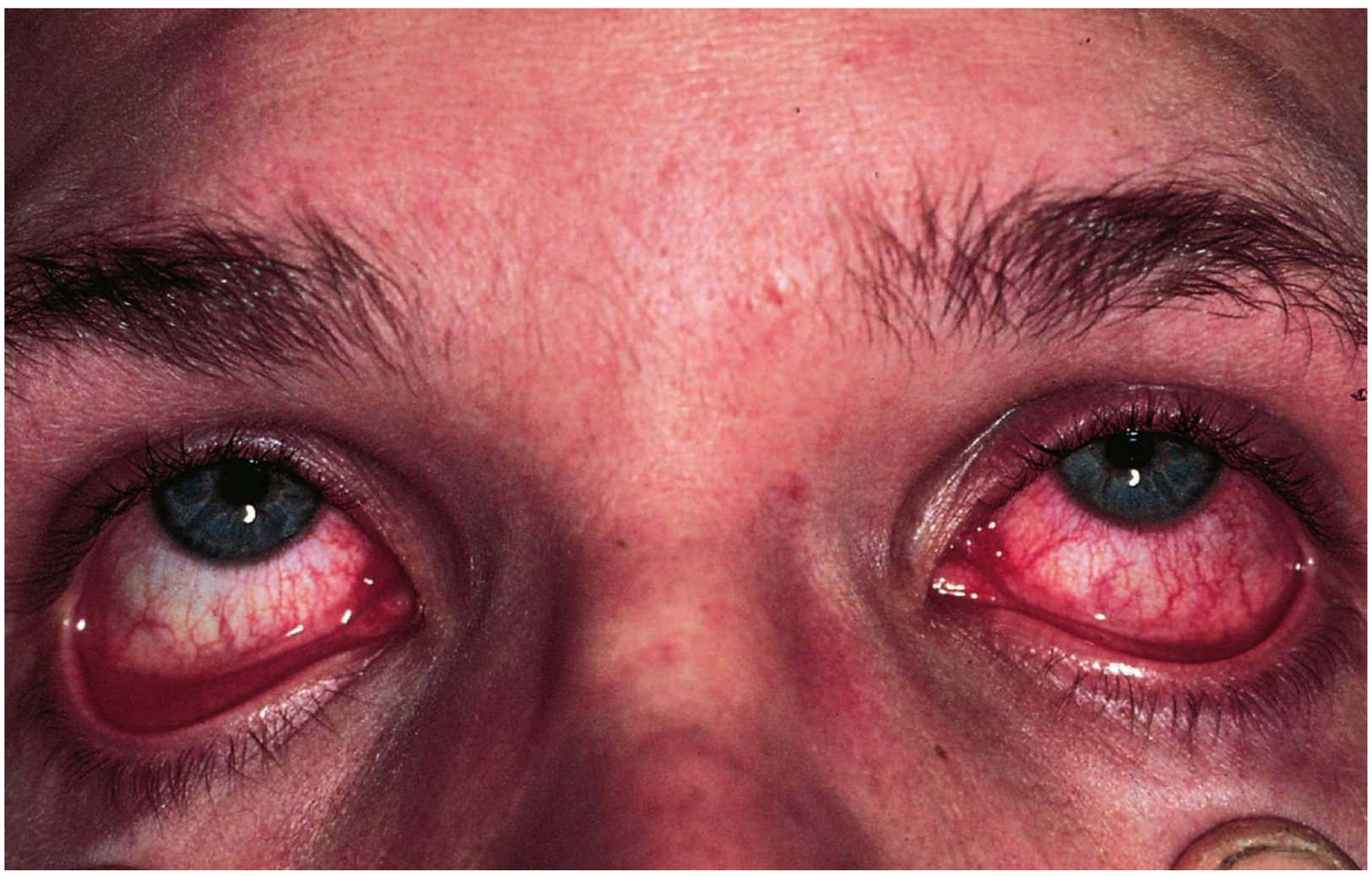

FIGURE 2.8 ■ **Viral Conjunctivitis.** Note the characteristic asymmetric conjunctival injection. Symptoms first developed in the left eye, with symptoms spreading to the other eye a few days later. A thin watery discharge is also seen. (Photo contributor: Kevin J. Knoop, MD, MS.)

FIGURE 2.9 ■ **Epidemic Keratoconjunctivitis (EKC).** Diffuse injection of the bulbar conjunctiva is seen in addition to a papillary reaction of the palpebral conjunctiva—a classic finding in EKC. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

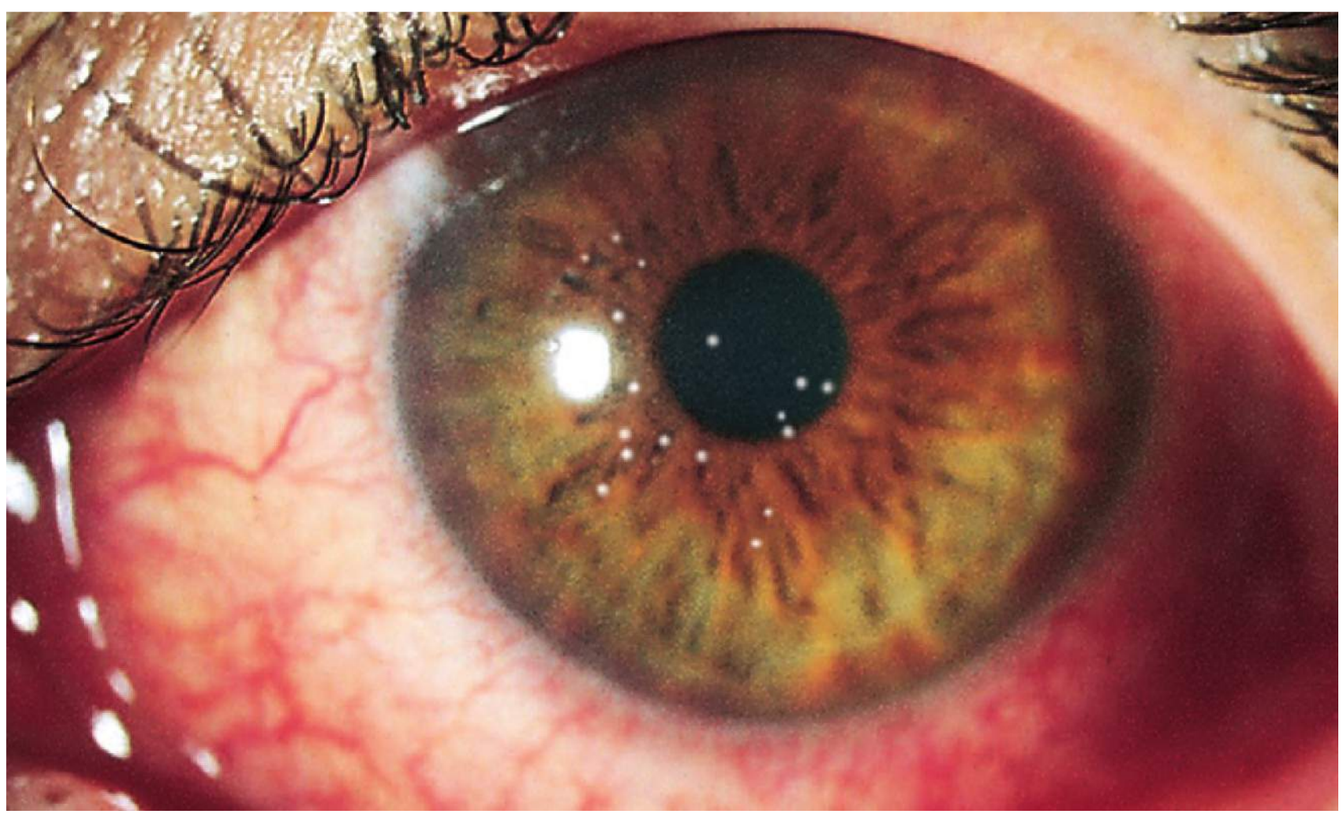

FIGURE 2.10 ■ **Subepithelial Infiltrates (EKC).** These usually develop in the second week. (Photo contributor: Katrina C. Santos, MD.)

Clinical Summary

Allergic conjunctivitis is a condition whereby airborne allergens precipitate type 1 IgE-mediated hypersensitivity reactions in the conjunctiva. Allergens include pollens, animal dander, mites, mold, and dust. Approximately 50% of patients have a personal or family history of allergic conditions such as atopy, eczema, asthma, and allergic rhinitis.

Itching is the hallmark symptom. Associated clinical features include conjunctival injection and edema, burning, discharge (clear, white, or mucopurulent), chemosis, and eyelid redness and swelling. Small papillae may be seen on the tarsal conjunctiva.

Vernal conjunctivitis is a rare but serious form of allergic conjunctivitis. The highest incidence is seen in the arid areas of the Middle East and North Africa secondary to wind and dust storms. Symptoms are similar to allergic conjunctivitis, but are more intense. Itching is severe and a vigorous knuckle rubbing is a typical observation. Giant, raised, pleomorphic papillae ("cobblestones") seen over the upper tarsal plate are pathognomonic.

Management and Disposition

The initial approach is to eliminate the allergen. Avoiding animal dander, using air conditioners with appropriate filters, and limiting time outdoors will improve the condition. Topical tear substitutes are effective to dilute or wash away the allergen. H1 antihistamine-vasoconstrictor combinations such as over-the-counter Naphcon A are recommended to relieve mild itching and redness. For more severe or frequent attacks, olopatadine, an antihistamine with mast cell stabilizing properties, is more effective. Mild topical steroids are an option only after consultation with an ophthalmologist.

Options for vernal conjunctivitis include cromolyn, aspirin, and cold compresses. Topical cyclosporin may be useful in resistant cases.

Pearls

1. Itching is the hallmark symptom of allergic conjunctivitis.
2. Itching is not common in nonallergic conjunctivitis.
3. Symptoms are usually bilateral.
4. Naphcon A and olopatadine are effective in treating allergic conjunctivitis.
5. Complications of topical corticosteroid use include glaucoma, cataract formation, secondary infection, and corneal perforation.

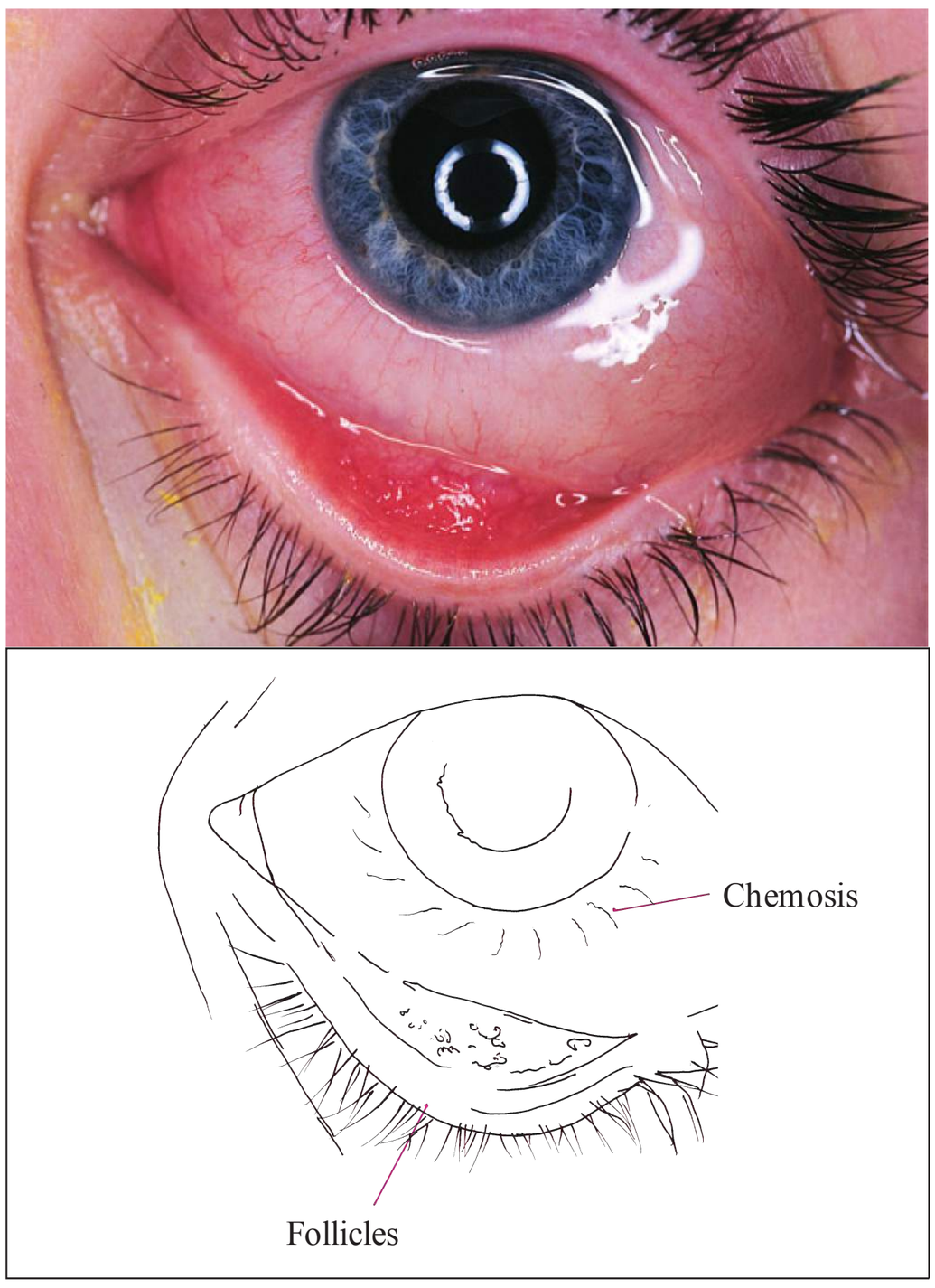

FIGURE 2.11 ■ **Allergic Conjunctivitis.** Conjunctival injection, chemosis, and a follicular response in the inferior palpebral conjunctiva are seen in this patient with allergic conjunctivitis secondary to cat fur. (Photo contributor: Timothy D. McGuirk, DO.)

FIGURE 2.12 ■ **Vernal Conjunctivitis.** The tarsal conjunctiva demonstrates giant papillae and a cobblestone appearance pathognomonic for vernal conjunctivitis. (Photo contributor: William Beck, CRA.)

DACRYOCYSTITIS

Clinical Summary

Dacryocystitis is an inflammation of the lacrimal sac, positioned immediately distal to the canaliculi and proximal to the nasolacrimal duct. Inflammation is usually secondary to obstruction of the nasolacrimal duct. Clinical findings include swelling over the lacrimal sac, redness, tearing, eyelash matting and crusting, and conjunctival redness. Tears and mucopurulence may be expressed from the punctum when pressure is applied over the lacrimal sac. Complications include conjunctivitis and orbital or preseptal cellulitis.

Up to 20% of normal newborns have a closed nasolacrimal passage, and 90% spontaneously open within the first 6 months.

Management and Disposition

Ophthalmologic consultation is recommended. The most common organisms isolated in children are S aureus, Staphylococcus epidermidis, and α-hemolytic Streptococci. Oral clindamycin for 7 to 10 days is recommended for outpatient management. Febrile and acutely ill patients require IV vancomycin in combination with a third-generation cephalosporin.

Treatment of nasolacrimal duct obstruction is managed initially with downward lacrimal sac massage ("Crigler" massage) 2 to 3 times a day. Unresolved nasolacrimal duct obstruction requires lacrimal duct probing by the ophthalmologist.

Pearls

1. Nasolacrimal duct obstruction is the most common cause of persistent tearing and ocular discharge in children.
2. Swelling is localized to the extreme nasal aspect of the lower lid and may be confused with a hordeolum. Occasionally, conjunctival redness may be present.
3. Dacryocystitis may be confirmed by pressure on the lacrimal sac and the reflux of tears and purulent material from the punctum. The lacrimal sac and lacrimal fossa lie in the inferior medial aspect of the orbit, not on the side of the nose.
4. Urgent referral should be made for any signs of orbital cellulitis. These include proptosis, limitation of extraocular movements, and loss of vision.

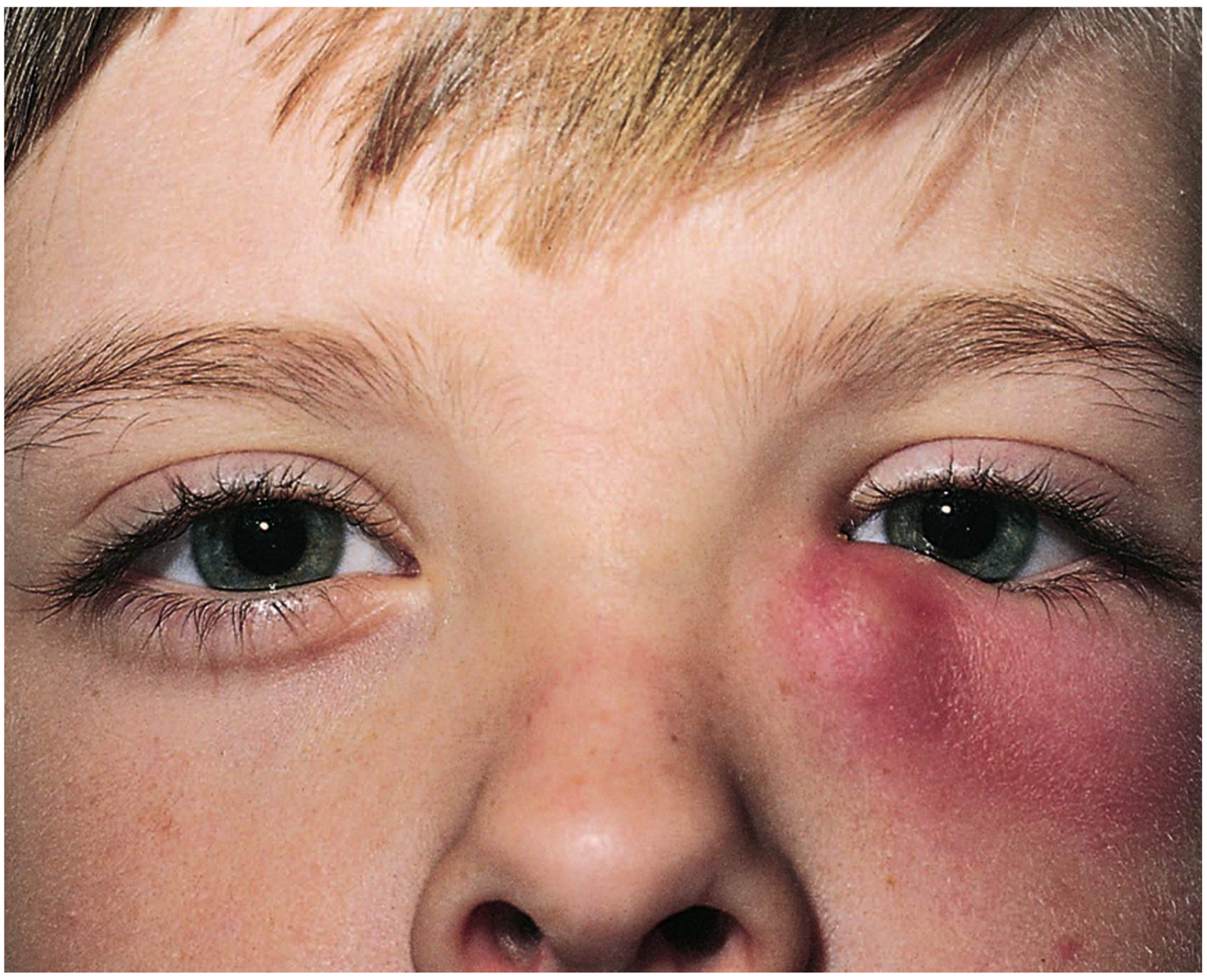

FIGURE 2.13 ■ **Dacryocystitis.** Swelling and erythema over the medial lower lid and lacrimal sac developed in this 10-year-old patient with streptococcal pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Acute dacryoadenitis typically involves children and young adults with associated systemic infections such as gonorrhea, mumps, Epstein-Barr virus, and Staphlyococcus species. Findings are localized to the outer one-third of the upper eyelid and include fullness or swelling, erythema, and tenderness. A characteristic "S"-shaped deformity with ptosis of the lid may be seen. In more advanced cases proptosis, inferonasal globe displacement, ophthalmoparesis, and diplopia may be present.

Chronic dacryoadenitis is more common, is seen in older patients, and is usually due to tumor or associated inflammatory disorders such as sarcoidosis, Sjögren syndrome, or IgG4-related diseases.

Management and Disposition

For acute dacryoadenitis, amoxicillin-clavulanate or IV ampicillin-sulbactam are used, depending on the severity and patient's toxicity. In cases of dacryoadenitis due to mumps or Epstein-Barr virus, warm compresses are recommended. Resolution occurs spontaneously. Patients should return to the ED for symptoms suggestive of orbital cellulitis such as decreased ocular motility or proptosis.

Treatment of chronic dacryoadenitis involves treatment of the underlying disorder.

Nonemergent ophthalmology follow-up is appropriate.

Pearls

1. Swelling is localized over the lateral one-third of the upper lid and imparts an "S"-shaped curve to the lid margin.
2. Acute dacryoadenitis is usually seen as a complication of mumps, with (bilateral) parotid swelling.
3. Chronic dacryoadenitis is more common, and is seen in older patients. Malignancy should be considered.
4. IgG4-related disease should be considered in patients (particularly middle-aged and older men) with bilateral disease and either salivary gland enlargement or pancreatitis of unknown origin.
5. Urgent referral is recommended for patients with diplopia, limitation of the extraocular muscles, or reduction of vision.

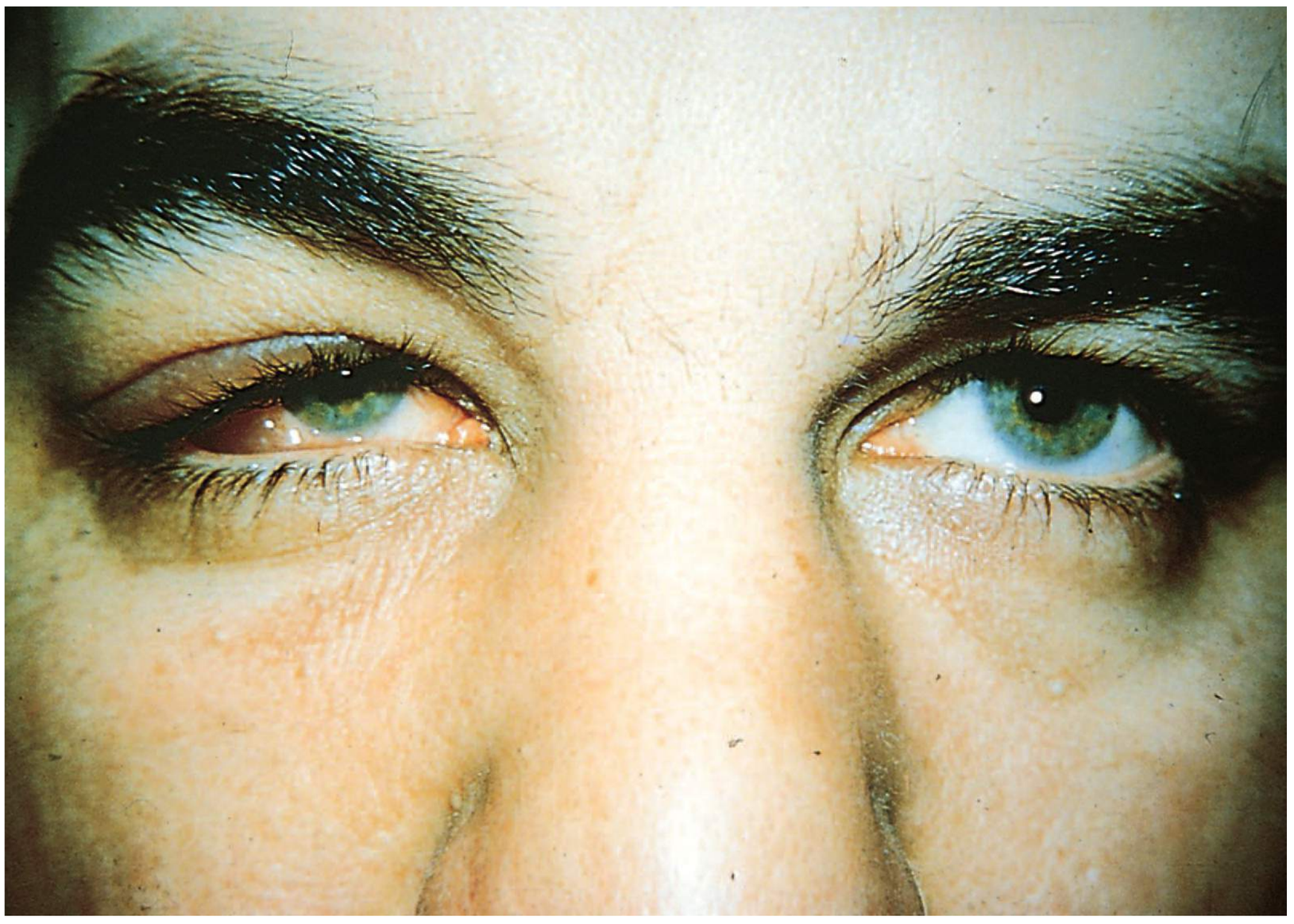

FIGURE 2.14 ■ **Dacryoadenitis.** Unilateral localized swelling and chemosis are present laterally secondary to inflammation of the lacrimal gland. (Used with permission from the American Academy of Ophthalmology. External Disease and Cornea: A Multimedia Collection. San Francisco, 1994.)

PTERYGIUM/PINGUECULA

Clinical Summary

Pterygium (Greek, pterygion meaning wing-like) is a benign proliferation of fibrovascular tissue. It usually originates in the nasal conjunctiva on the horizontal meridian of the limbus. It progressively encroaches onto the cornea and visual axis. Its appearance ranges from flat and white to thick, pink or red, and fibrovascular. Typically, it assumes a triangular appearance, the apex directed toward the pupil. Predisposing factors include exposure to ultraviolet light, wind, and dust. Older individuals living in warmer areas with high levels of sunlight are more likely to develop pterygia.

Pterygia may be asymptomatic or become inflamed, causing mild symptoms of irritation and foreign body sensation. Decreased visual acuity may result as the pterygium encroaches on the visual axis or if the lesion induces astigmatism.

Pinguecula (Latin, pingueculus meaning fatty) is a common degenerative lesion of the bulbar conjunctiva, also arising in the horizontal meridian. It appears as a light brown or yellow-white amorphous, slightly raised conjunctival tissue adjacent to the limbus. It too may be asymptomatic or become episodically inflamed; however, it may enlarge and become a pterygium.

Management and Disposition

Mild disease can be treated with artificial tears or a topical vasoconstrictor (Naphcon A). In more severe cases, consultation with an ophthalmologist is appropriate regarding topical steroids and topical nonsteroidal anti-inflammatory drugs (NSAIDs). Surgical excision by the ophthalmologist is indicated if the pterygium interferes with contact lens wear, encroaches significantly on the visual axis resulting in induced astigmatism or opacity, or restricts eye movement.

Pearls

1. Pterygia are more likely than pinguecula to be found on the nasal conjunctiva and bilateral.
2. Pterygia and pingueculae are found on the horizontal meridian. Conjunctival neoplasms often occur in axes other than the horizontal axis.
3. Following surgical excision, recurrence of pterygia is common.
4. Pterygium-mimics include localized conjunctival neoplasia, conjunctivitis, and episcleritis.

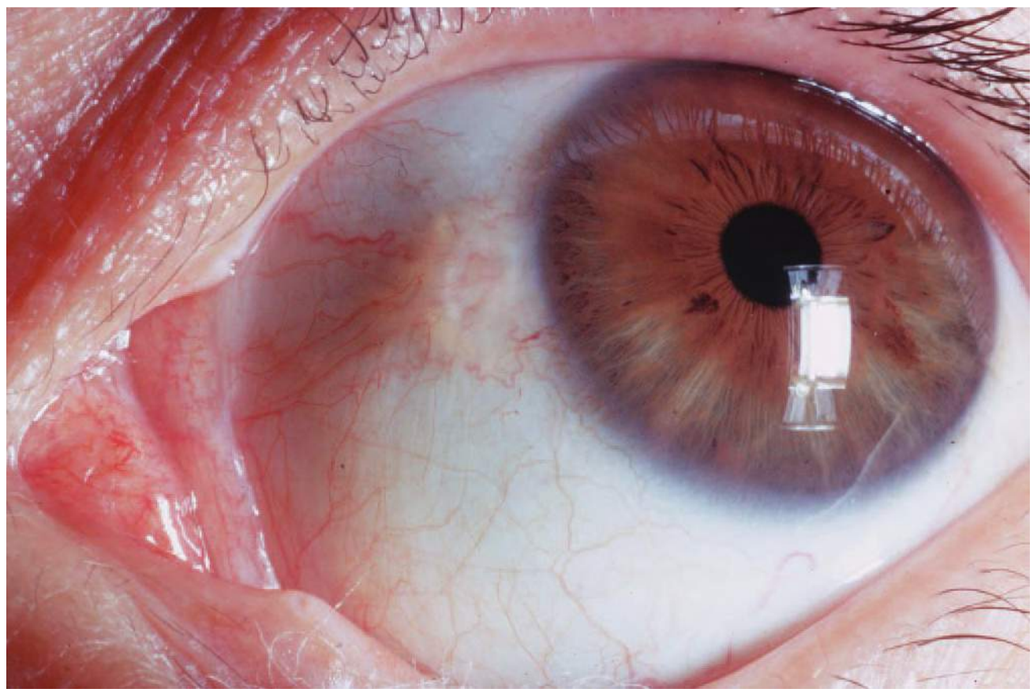

FIGURE 2.15 ■ **Pinguecula.** A small area of yellowish "heaped up" conjunctival tissue is seen adjacent to the nasal limbus. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

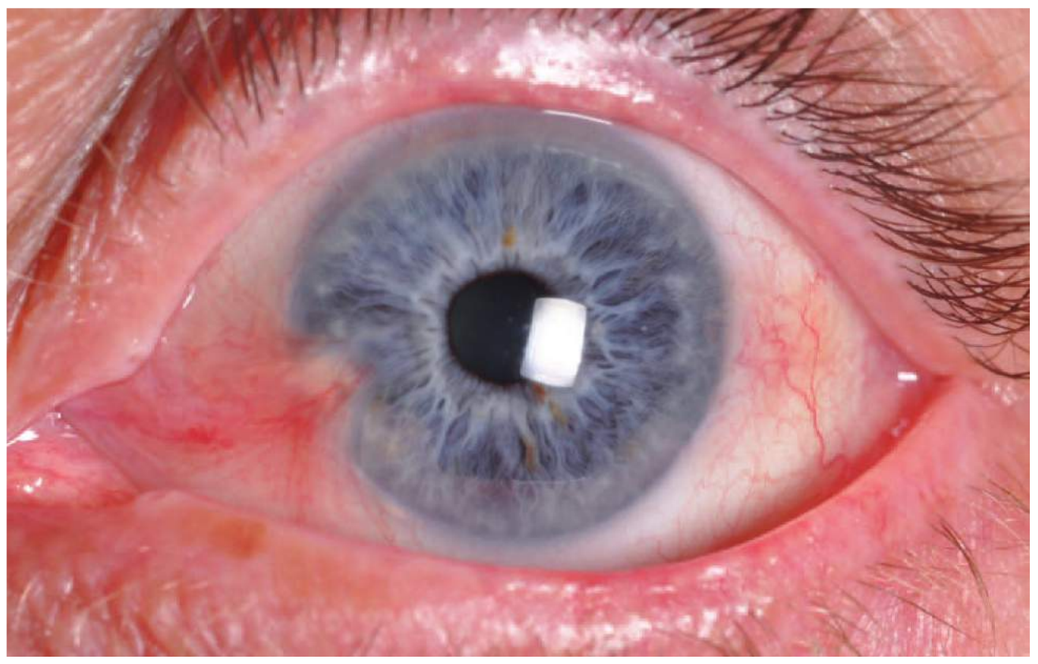

FIGURE 2.16 ■ **Pterygium.** Pterygium appears as a raised vascular triangular area of bulbar conjunctiva that encroaches onto the cornea. (Photo contributors: Top: Rebecca Kasl, MD; Bottom: Andrew J. Hendershot, MD.)

Clinical Summary

A hordeolum is a localized abscess involving the glands of the eyelid. An external hordeolum (stye) involves an eyelash (cilia) follicle or the tear glands of the lid margin (glands of Zeis, Moll). An internal hordeolum involves the meibomian glands. S aureus is the most frequent isolate. Clinical findings include focal swelling, edema, erythema, and tenderness.

A chalazion is a chronic localized inflammation that results from the obstruction of the meibomian glands. It may arise from a hordeolum. The lesion may point anteriorly (toward the skin of the eyelid) or posteriorly (toward the tarsal conjunctiva). It may become sufficiently large as to press on the globe and cause astigmatism. Pain and redness may be seen early in the course, but these may dissipate over time.

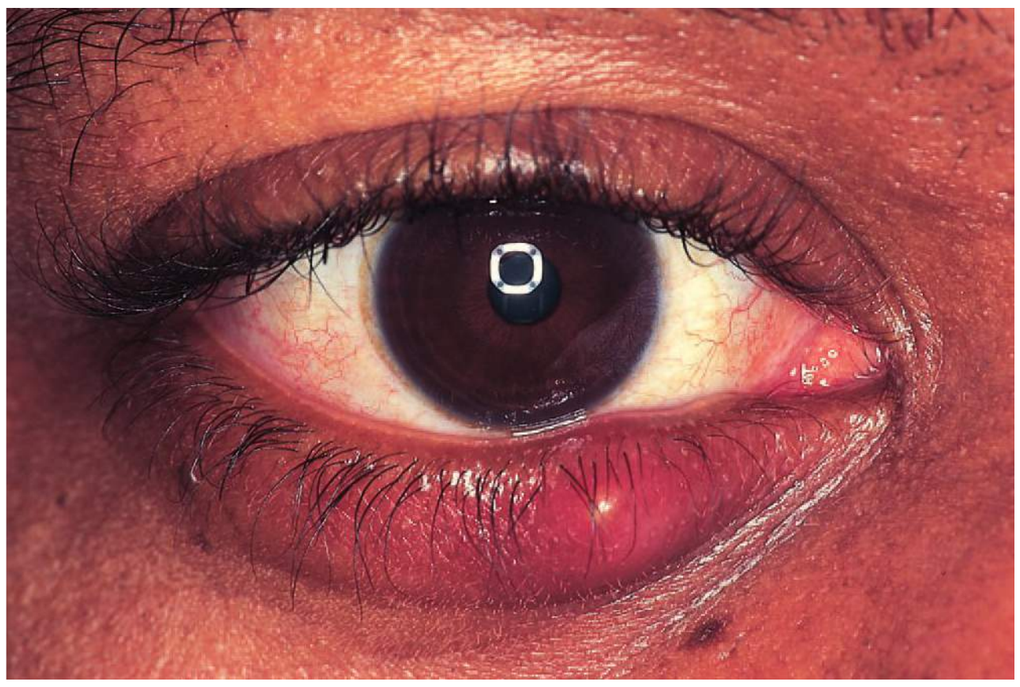

FIGURE 2.18 ■ **Hordeolum.** Focal swelling and erythema at the lid margin are seen in this hordeolum. (Photo contributor: Frank Birinyi, MD.)

Management and Disposition

Hordeola can be treated with warm compresses. The clinical course of a hordeolum is usually self-limited, with spontaneous drainage and resolution in 5 to 7 days. Topical antibiotics are not helpful. Systemic antibiotics are unnecessary unless there is concurrent preseptal cellulitis. If the mass persists or is large (distorting vision), ophthalmology referral is recommended for incision and curettage or intralesional corticosteroid injection.

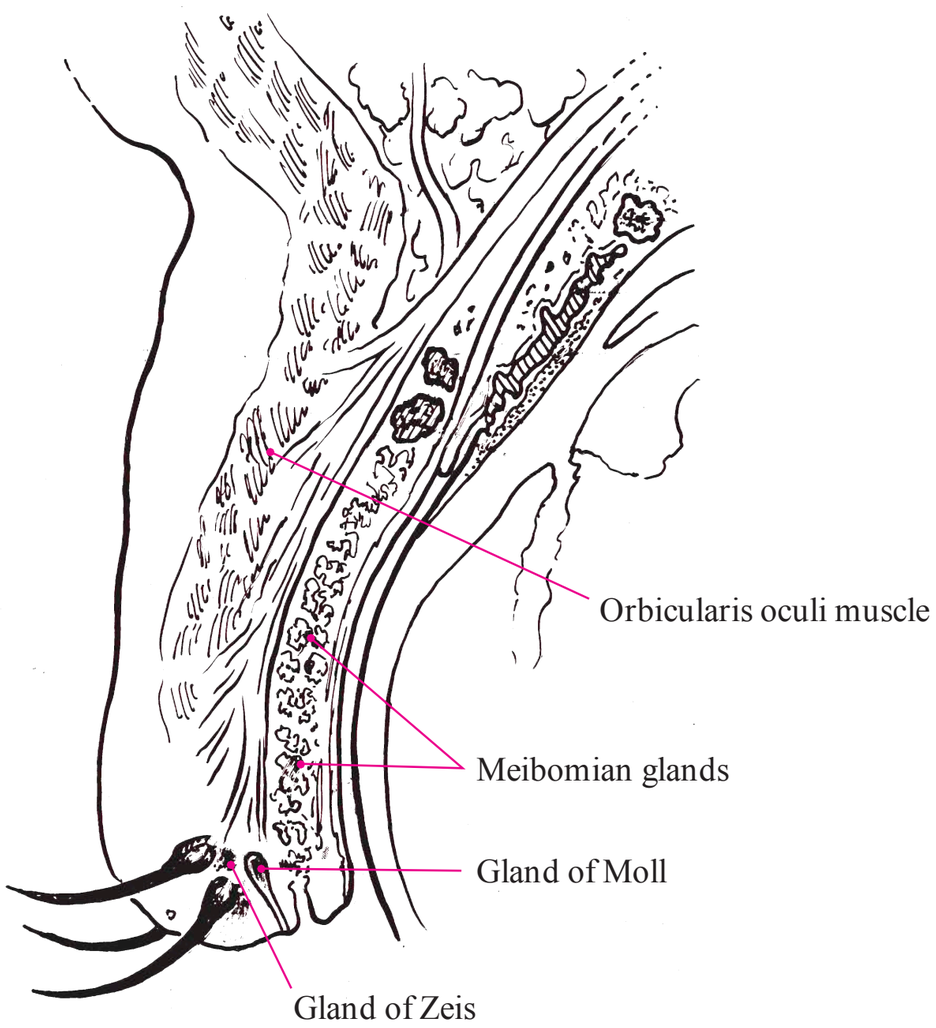

FIGURE 2.17 ■ **Eyelid Anatomy.** Anatomic structures related to eyelid pathology.

Frequent warm compresses applied to chalazia expedite spontaneous drainage. Most chalazia spontaneously clear after several weeks. Antibiotics are not indicated. Recalcitrant lesions should be referred to the ophthalmologist for incision and curettage or glucocorticoid injection.

Pearls

1. Uncomplicated hordeola and chalazia can be treated with warm compresses, and generally resolve spontaneously.
2. Patients with rosacea or marginal blepharitis (a chronic low-grade inflammation of the lid margins with crusts around the lashes) have frequent hordeola.
3. Older patients with recurrent chalazia should be referred to the ophthalmologist to rule out malignancy.

FIGURE 2.19 ■ **Chalazion.** This chalazion shows painful nodular focal swelling and erythema from meibomian cyst formation. (Photo contributor: Frank Birinyi, MD.)

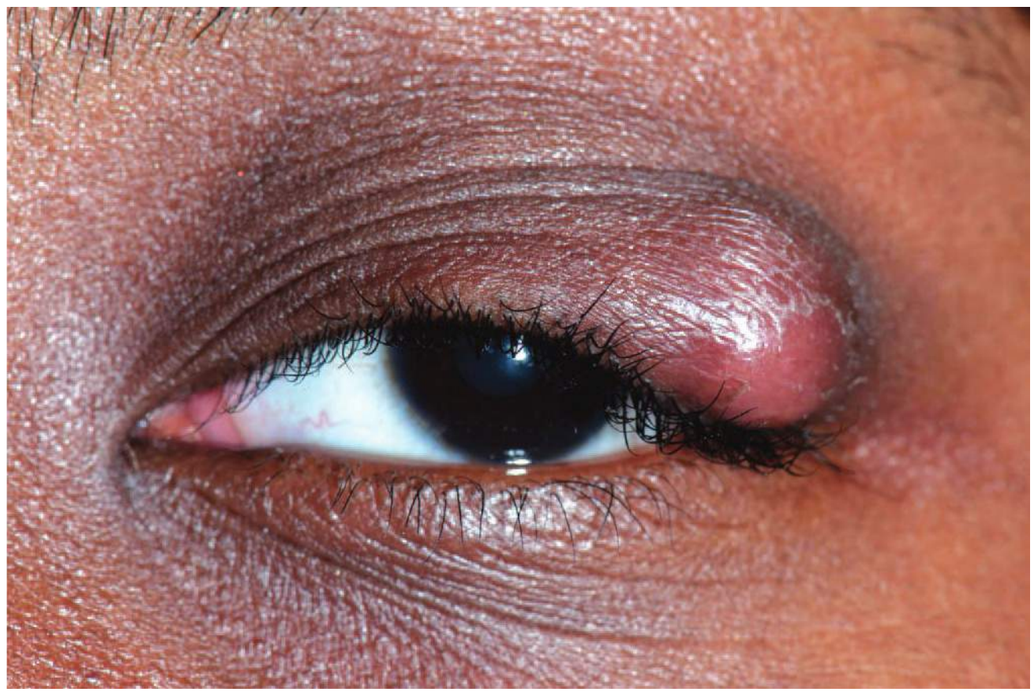

FIGURE 2.20 ■ **Chalazion.** Two months of focal pain and swelling of the lateral upper lid. (Photo contributor: Lawrence B. Stack, MD.)

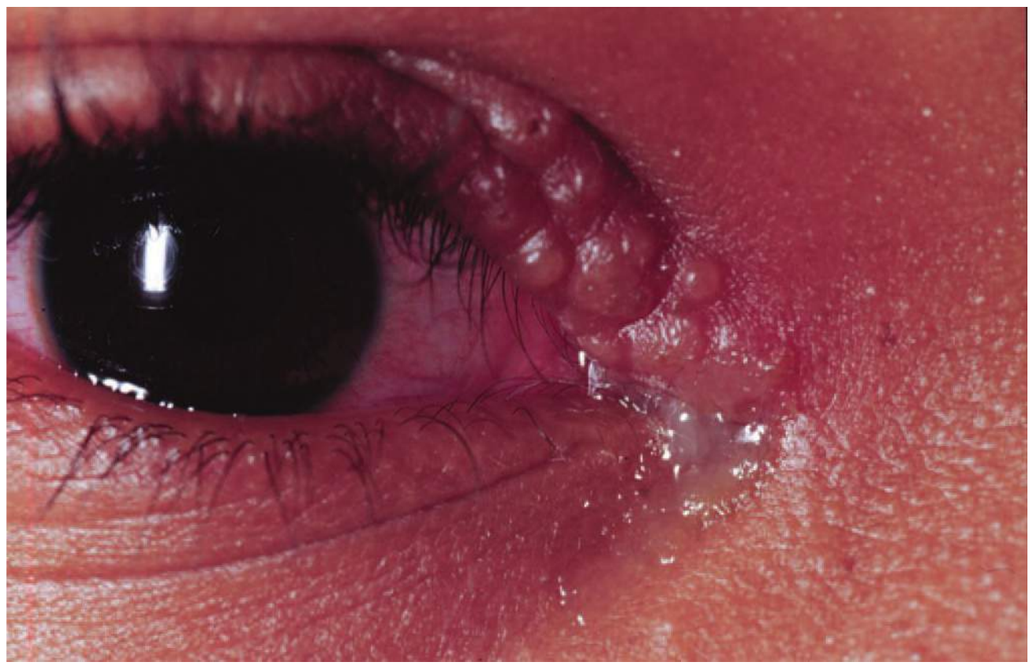

FIGURE 2.22 ■ **Ocular Herpes Simplex.** This 10-year-old has recurrent ocular herpes simplex since age 6. Vesicles should not be mistaken for hordeola. (Photo contributor: Lawrence B. Stack, MD.)

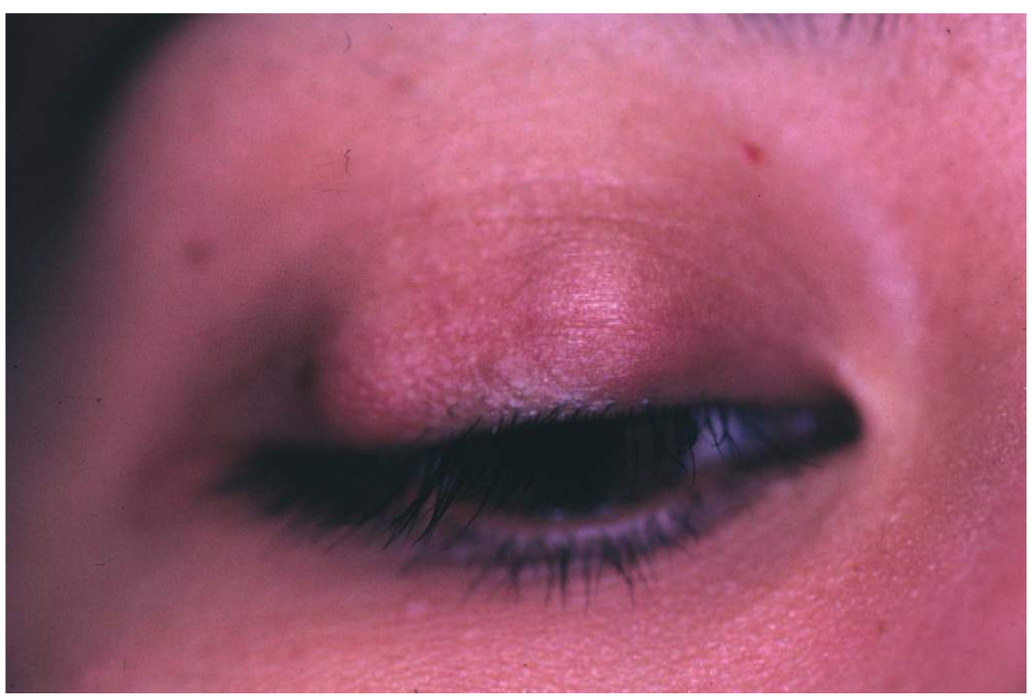

FIGURE 2.21 ■ **Chalazion.** A nontender bipartite chalazion is shown. (Photo contributor: Kevin J. Knoop, MD, MS.)

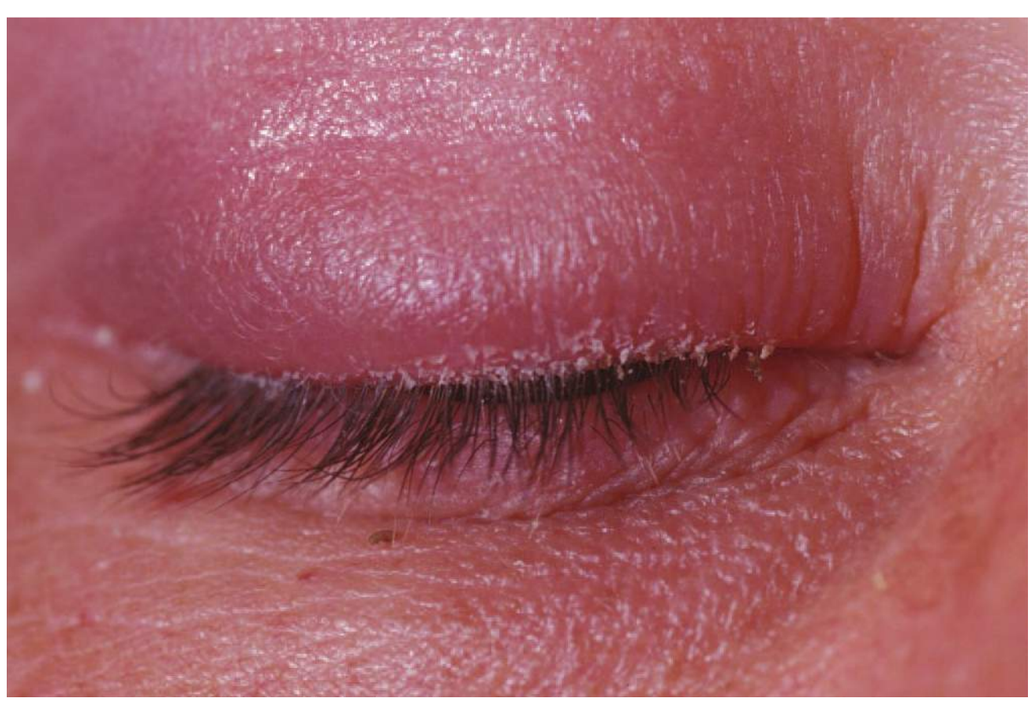

FIGURE 2.23 ■ **Blepharitis.** The eyelid margins are inflamed and erythematous. (Photo contributor: Kevin J. Knoop, MD, MS.)

FIGURE 2.24 ■ **Chalazion.** This lower lid chalazion was seen only with lid eversion. (Photo contributor: James Dahle, MD.)

Clinical Summary

Scleritis is painful, destructive, and potentially blinding. The pain is constant and boring and may radiate to the face and periorbital region. Associated features include tearing, photophobia, globe tenderness to palpation, and painful ocular movement.

The conjunctival vessels are injected. The eye itself may be intensely red with a violaceous or purple hue secondary to engorgement of the deep vessels of the episclera and scleral thinning. These deep vessels do not move when the overlying conjunctiva is moved with a cotton-tipped applicator, nor do they blanch with topical phenylephrine. On slit-lamp microscopy, the episcleral vessels are displaced outward by scleral edema. Corneal involvement, iritis (with cells and flare in the anterior chamber), and decreased visual acuity may accompany scleritis. An associated scleritis may occur with severe infectious keratitis. Primary infectious scleritis is rare. In either instance, topical and systemic antibiotics are indicated after appropriate cultures are obtained.

In up to 50% of cases, scleritis is associated with underlying autoimmune or infectious systemic disease, rheumatoid arthritis the most common. It occurs more frequently in women and in the fourth to sixth decades of life.

Management and Disposition

Ophthalmology consultation is required. Treatment varies according to underlying disease (if present), and can involve NSAID therapy, glucocorticoids, and immunosuppressive medications. Rheumatology consultation by the ophthalmologist is often required for optimal management in patients with underlying autoimmune disorders.

Pearls

1. Scleritis is associated with a systemic disease in approximately 50% of cases, most commonly rheumatoid arthritis.
2. Most cases of scleritis involve the anterior portion (anterior to the insertion of the medial and lateral rectus muscles).
3. Pain is exacerbated with ocular movements because the extraocular muscles insert into the sclera itself.
4. Anterior uveitis can occur in up to 40% of patients because the uvea is immediately adjacent to the sclera.
5. Check intraocular pressure (IOP) to rule out acute ACG as another cause of painful red eye.

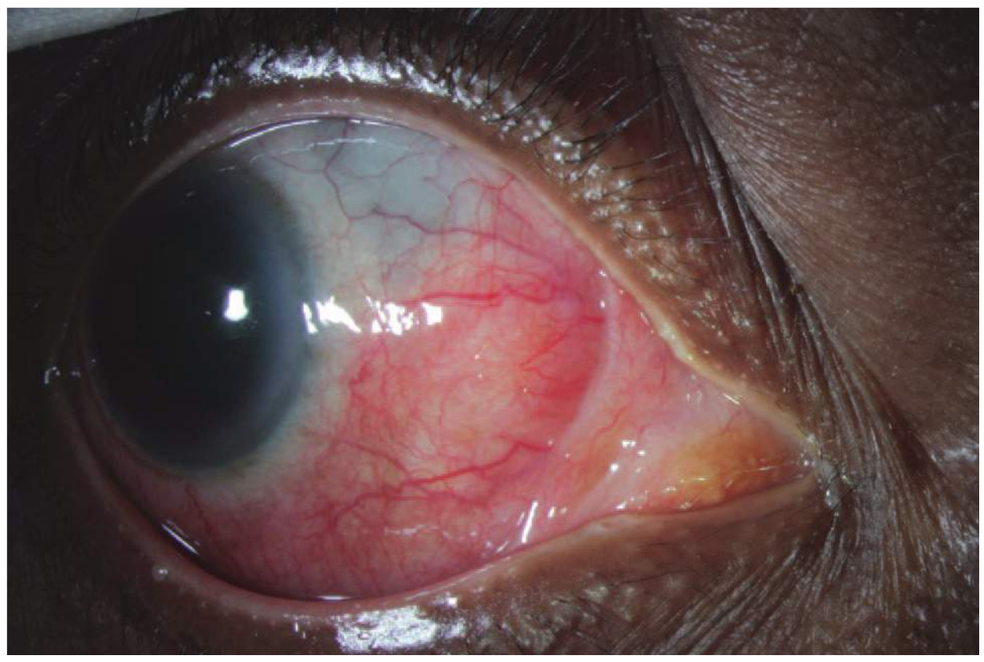

FIGURE 2.25 ■ **Scleritis.** A prominent generalized vascular injection is present. A bluish hue is also seen superiorly due to scleral thinning. These vessels do not move when the overlying conjunctiva is moved with a cotton-tipped applicator. (Photo contributor: Jeffrey Goshe, MD.)

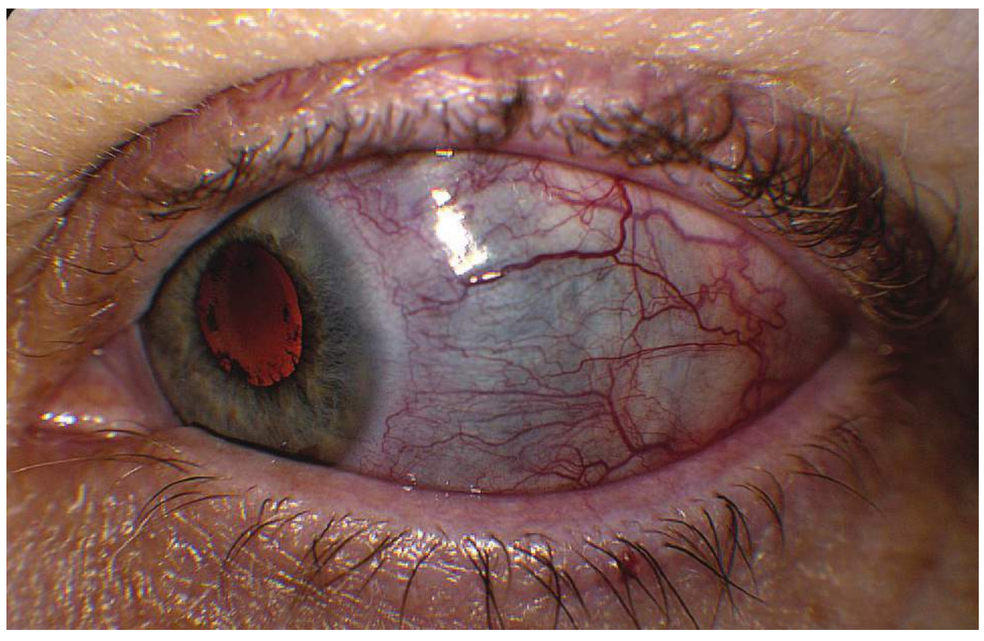

FIGURE 2.26 ■ **Scleritis.** A 55-year-old female with scleritis of the left eye associated with rheumatoid arthritis. Note dilation of the deep conjunctival and episcleral vessels and blue hue suggesting thinning of the sclera temporally. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

FIGURE 2.27 ■ **Sectorial Scleritis.** Deep-boring pain experienced by this patient distinguishes this segmental area of erythema from episcleritis. (Photo contributor: Kevin J. Knoop, MD, MS.)

EPISCLERITIS

Clinical Summary

Episcleritis is a common and benign inflammation of the episclera, typically affecting young and middle-aged adults. Seventy percent of cases occur in females. The episclera lies just beneath the bulbar conjunctiva. Its vessels are large, run in a radial direction, and can be seen beneath the overlying conjunctiva. Episcleral and conjunctival vessels blanch with the use of topical 5% phenylephrine drops, unlike deep episcleral vessels.

Patients may complain of foreign body sensation, mild tenderness, irritation, mild photophobia, and excessive lacrimation. Pain is unusual but can occur, particularly in chronic cases. One-half of cases are bilateral. Eye findings are notable for a localized pink or bright red conjunctival injection, with involvement of the vessels in the superficial episcleral vascular plexus. Visual acuity is normal.

Episcleritis is usually an isolated condition, though it may be associated with a number of systemic diseases, including rheumatoid arthritis, inflammatory bowel disease, lupus, and vasculitis.

Management and Disposition

For many patients, the condition is self-limited and will resolve within 3 weeks, with or without treatment. For mild cases, use over-the-counter artificial tears. For those with recurrent or recalcitrant lesions, referral to the ophthalmologist is indicated.

Pearls

1. It is important to differentiate episcleritis from sight-threatening scleritis. Episcleritis presents with only mild pain and bright red episcleral vessels, which will blanch with topical phenylephrine.
2. Conjunctivitis is a more common cause of red eye, and symptoms usually include morning crusting. Injection that is localized rather than diffuse is more likely to be episcleritis.

FIGURE 2.28 ■ **Episcleritis.** A localized area of hyperemia is seen involving the temporal sclera. (Photo contributor: Robert Trieff, MD.)

Clinical Summary

Acute angle-closure glaucoma (ACG) is secondary to narrowing or closure of the anterior chamber angle, resulting in increased IOP, with subsequent damage to the optic nerve. Normally, aqueous humor drains out of the anterior chamber via Schlemm canal in the anterior chamber angle. In ACG, this flow is impeded, and the IOP rises from a normal range of 10 to 21 mm Hg to 50 mm Hg or higher.

ACG presents as an acutely inflamed eye. Nausea and vomiting are common and may be the presenting complaints. Eye pain and headache vary in severity. As the IOP reaches the 50 to 60 mm Hg range, fluid is forced into the cornea, resulting in corneal edema. Patients report blurred vision and rainbow-colored halos around lights. Clinical findings include tearing, perilimbal injection ("ciliary flush"), a cloudy ("steamy") cornea, a nonreactive mid-dilated pupil, anterior chamber inflammation, and an increased IOP. Using a penlight or slit-lamp microscopy, the anterior chamber may appear shallow.

Management and Disposition

ACG is an emergency that requires ophthalmology consultation, and treatment is directed at reducing the IOP. Aqueous outflow is increased by topical myotics (pilocarpine) that pull the peripheral iris taut away from the anterior chamber angle. Decreased production of aqueous is accomplished with topical β-blockers (timolol), an α-adrenergic agonist (apraclonidine), or a carbonic anhydrase inhibitor (acetazolamide or methazolamide systemically, dorzolamide topically). Osmotic agents reduce the aqueous volume within the eye and include glycerol, oral isosorbide, and IV mannitol. Latanoprost, a prostaglandin derivative, increases aqueous outflow through nontrabecular meshwork pathways.

The definitive treatment of ACG is laser peripheral iridectomy, done usually after the IOP normalizes.

Pearls

1. Vision loss and blindness can occur during the course of the attack (hours to days). ACG is a true ophthalmologic emergency.
2. Medicines with anticholinergic effects are capable of inducing pupillary dilation which might provoke an angle-closure attack. These include over-the-counter decongestants, motion sickness medications, antipsychotics, and antidepressants.
3. ACG often occurs in the evening, when lower light levels cause mydriasis.
4. ACG patients may easily be misdiagnosed initially as having a migraine headache or a CNS catastrophe because they may present with severe headache and vomiting.
5. The elevated IOP in acute ACG can be reduced pharmacologically by three mechanisms: first, by opening the closed angle with myotics; second, by reducing aqueous formation with β-blockers, α-agonists, or carbonic anhydrase inhibitors; and third, by reducing the aqueous volume within the eye by osmotic agents.

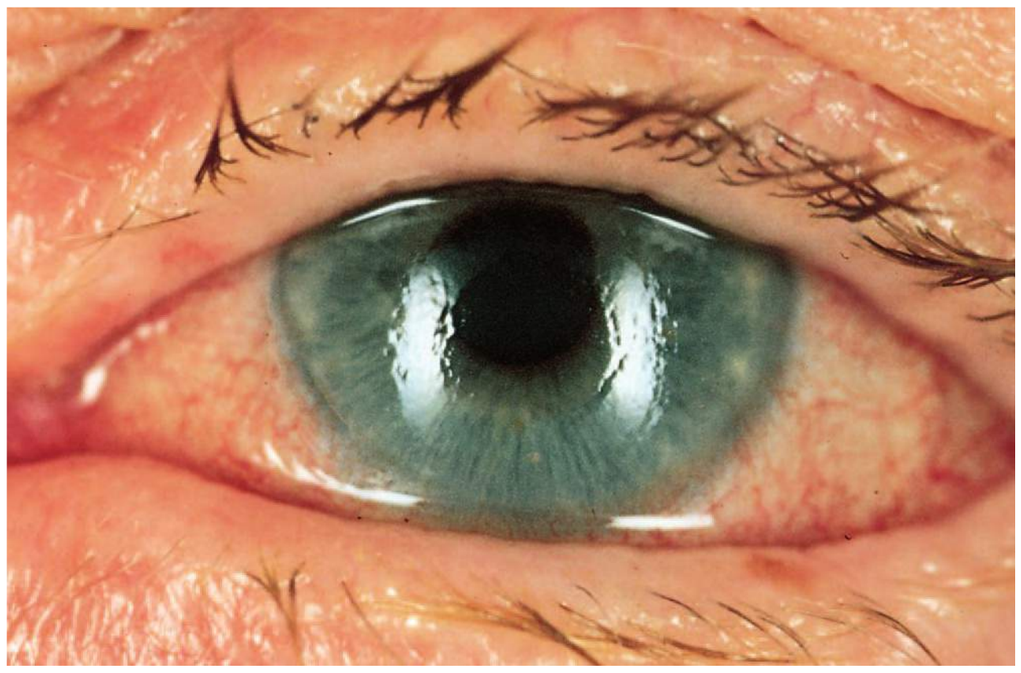

FIGURE 2.29 ■ **Acute Angle-Closure Glaucoma.** The cornea is edematous, manifest by the indistinctness of the iris markings and the irregular corneal light reflex. Conjunctival hyperemia is also present. (Photo contributor: Kevin J. Knoop, MD, MS.)

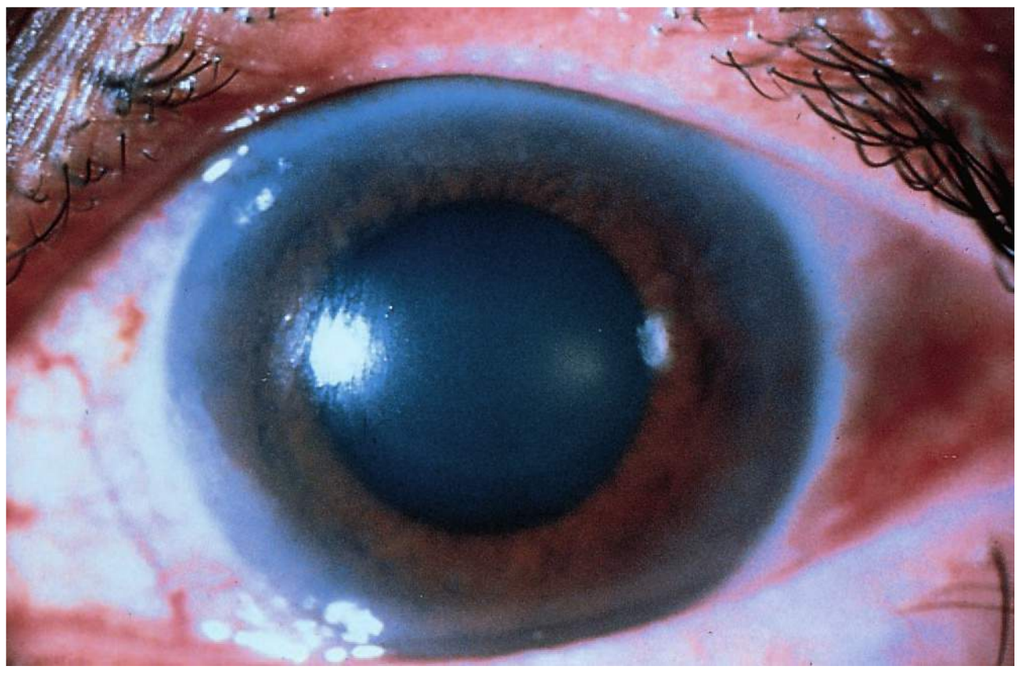

FIGURE 2.30 ■ **Acute Angle-Closure Glaucoma.** Note the cloudy or "steamy" appearance of the cornea and the midposition pupil. Conjunctival hyperemia is not as evident. (Photo contributor: Gary Tanner, MD.)

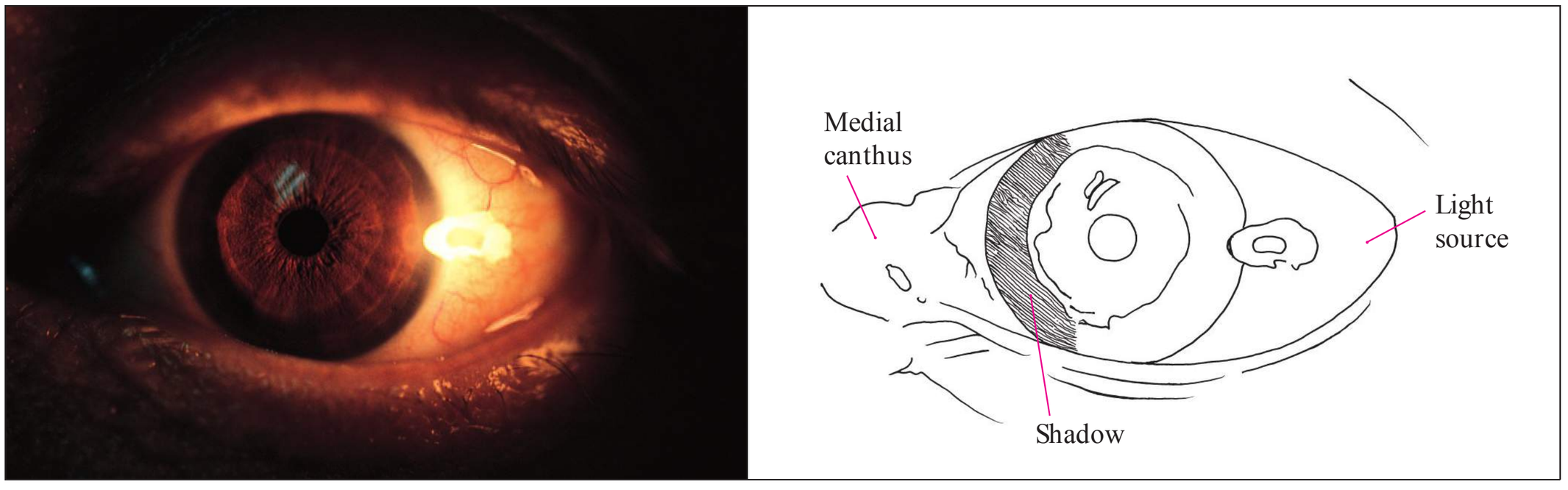

FIGURE 2.31 ■ **Penlight Test.** A penlight, held laterally and directed nasally, projects a shadow on the nasal side of an iris with a shallow anterior chamber. This patient presented with acute angle-closure glaucoma. (Photo contributor: Alan B. Storrow, MD.)

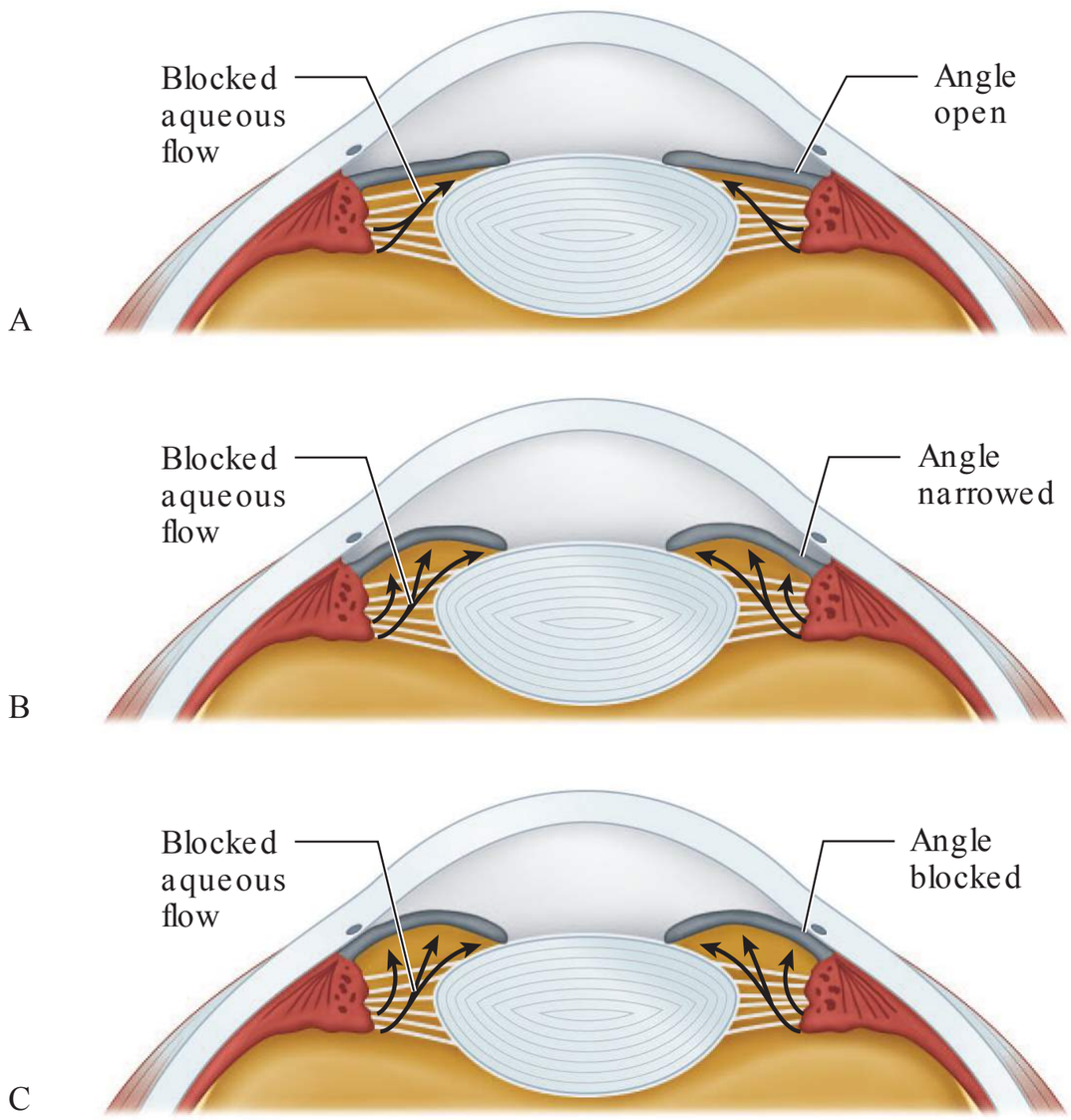

FIGURE 2.32 ■ **Iris Bombé.** (**A**) A physiologic block normally occurs where the posterior surface of the iris touches the lens. (**B**) When this block is increased, the iris as a result bows forward (iris bombé) and the angle is narrowed and pressure increases. (**C**) As pressure increases, the angle becomes blocked.

Clinical Summary

The uvea is the middle layer of the eye. Its anterior portion includes the iris and ciliary body; the posterior portion includes the choroid. Inflammation of the anterior portion is called anterior uveitis or iritis. It is often idiopathic, but approximately half of cases are associated with systemic disease. These include inflammatory disorders (rheumatoid arthritis, Behçet disease, sarcoid), HLA-B27-associated conditions (ankylosing spondylitis, inflammatory bowel disease, Reiter syndrome), and infectious causes (zoster, tuberculosis, toxoplasmosis, AIDS).

Clinical features include conjunctival hyperemia, hyperemic perilimbal vessels ("ciliary flush"), miosis, decreased visual acuity, photophobia, tearing, and pain. The slit-lamp may demonstrate a hypopyon, cells, flare, and keratic precipitates. Keratic precipitates are agglutinated inflammatory cells adherent to the posterior corneal endothelium. These precipitates appear either as fine gray-white deposits or as a large, flat, greasy-looking area ("mutton fat"). The IOP may be decreased due to decreased aqueous production by the inflamed ciliary body, or increased secondary to inflammatory debris within the trabeculae of the anterior chamber angle obstructing outflow.

Management and Disposition

The patient's history forms the basis for the evaluation and laboratory testing. The history should focus on rheumatic illness, dermatologic problems, bowel disease, infectious exposures, and sexual history. Treatment of the uveitis is nonspecific. Topical cycloplegics and corticosteroids may be prescribed in conjunction with the ophthalmologist. Antibiotics are not usually prescribed.

Pearls

1. Iritis is usually associated with a miotic pupil, pain, and redness primarily at the limbus ("ciliary flush").
2. When uveitis is associated with a systemic disorder, the associated condition is usually evident. Common exceptions include sarcoidosis and syphilis.
3. In patients with uveitis of unknown etiology, a chest x-ray looking for sarcoidosis and serologic testing for syphilis are reasonable.
4. Visual loss may occur with uveitis.
5. Topical analgesics do not significantly ameliorate the pain of anterior uveitis.
6. Consider sympathetic ophthalmia with unexplained uveitis and a history of eye trauma.

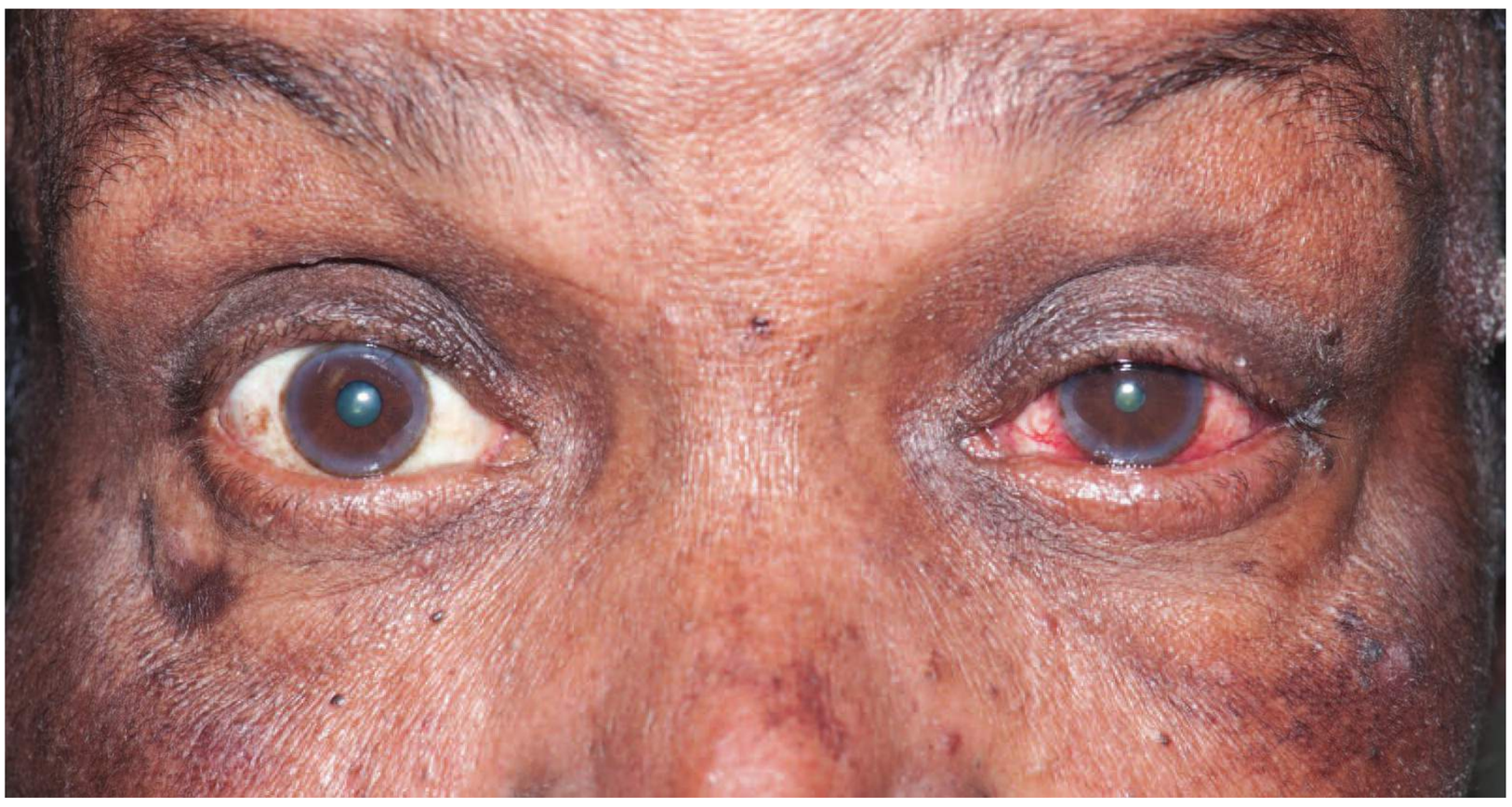

FIGURE 2.33 ■ **Anterior Uveitis**. Atraumatic left eye pain, photophobia, and limbal injection, in a middle-aged male with inflammatory bowel disease. Miosis is also present, a hallmark of uveitis. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 2.34 ■ **Anterior Uveitis**. Marked conjunctival injection and perilimbal hyperemia ("ciliary flush") are seen in this patient with recurrent iritis. (Photo contributor: Frank Birinyi, MD.)

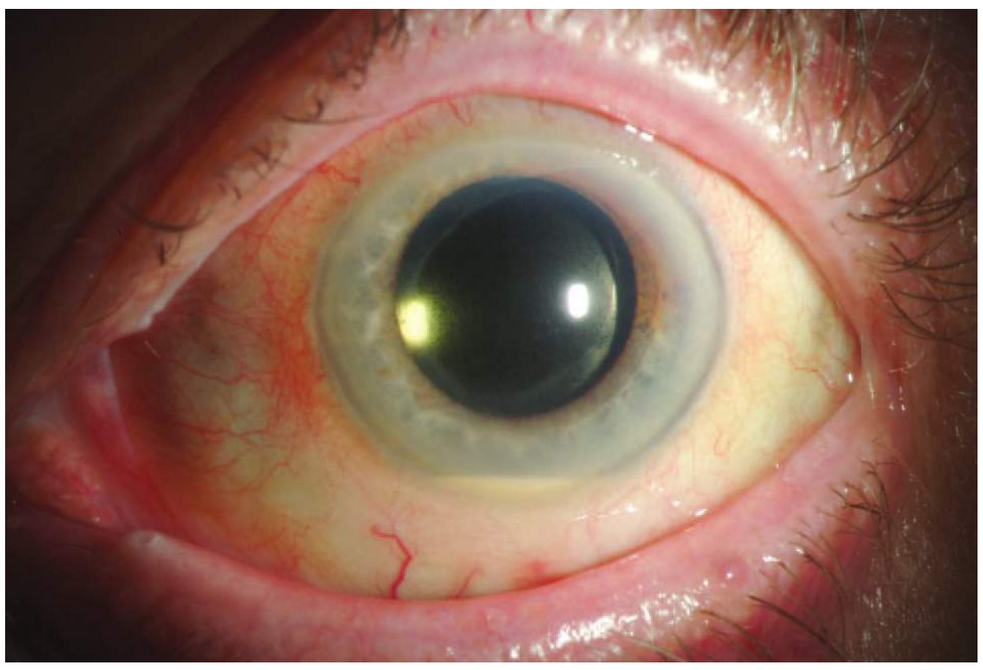

FIGURE 2.35 ■ **Hypopyon.** A thin layering of white blood cells is present in the inferior anterior chamber. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

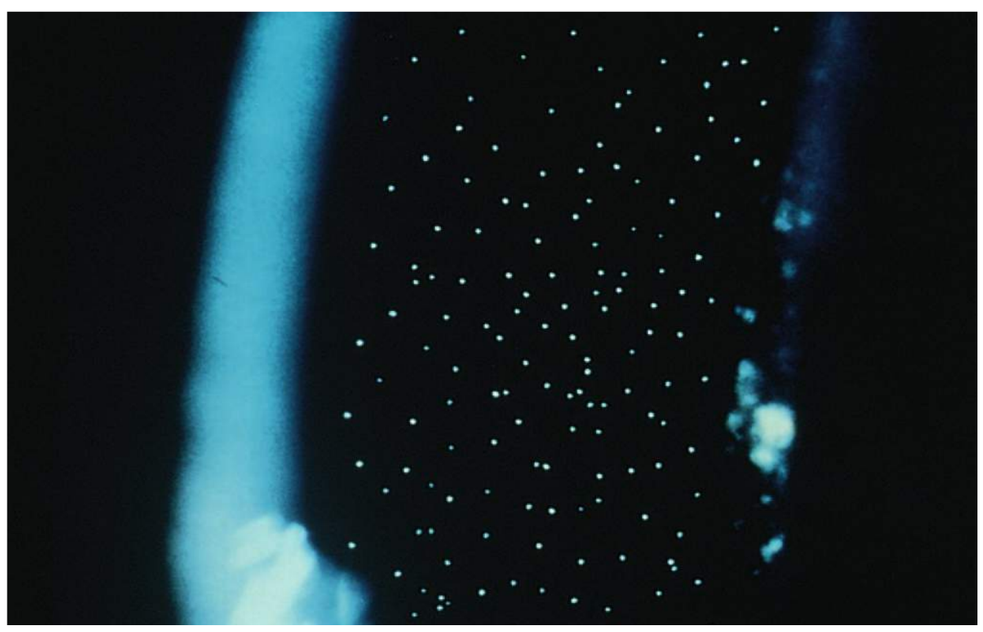

FIGURE 2.36 ■ **Anterior Chamber Cells**. Cells in the anterior chamber are a sign of inflammation or bleeding and appear similar to particles of dust in a sunbeam. They are best seen with a narrow slit-lamp beam directed obliquely across the anterior chamber. (Used with permission from Spalton DJ, Hitchings RA, Hunter PA (eds). Atlas of Clinical Ophthalmology. 2nd ed. London, UK: Mosby-Wolfe Limited; 1994.)

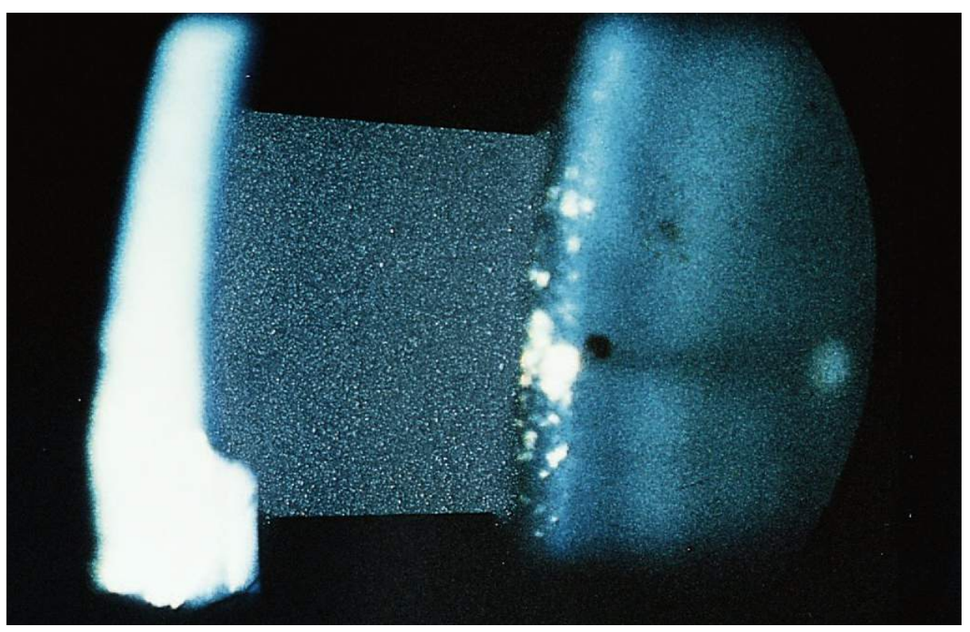

FIGURE 2.37 ■ **Anterior Chamber Flare.** Flare in the anterior chamber represents an elevated concentration of plasma proteins from inflamed, leaking intraocular blood vessels. Flare seen in a slit-lamp beam appears similar to a car headlight cutting through the fog. (Used with permission from Spalton DJ, Hitchings RA, Hunter PA (eds). Atlas of Clinical Ophthalmology. London, UK: Mosby-Wolfe Limited; 1984.)

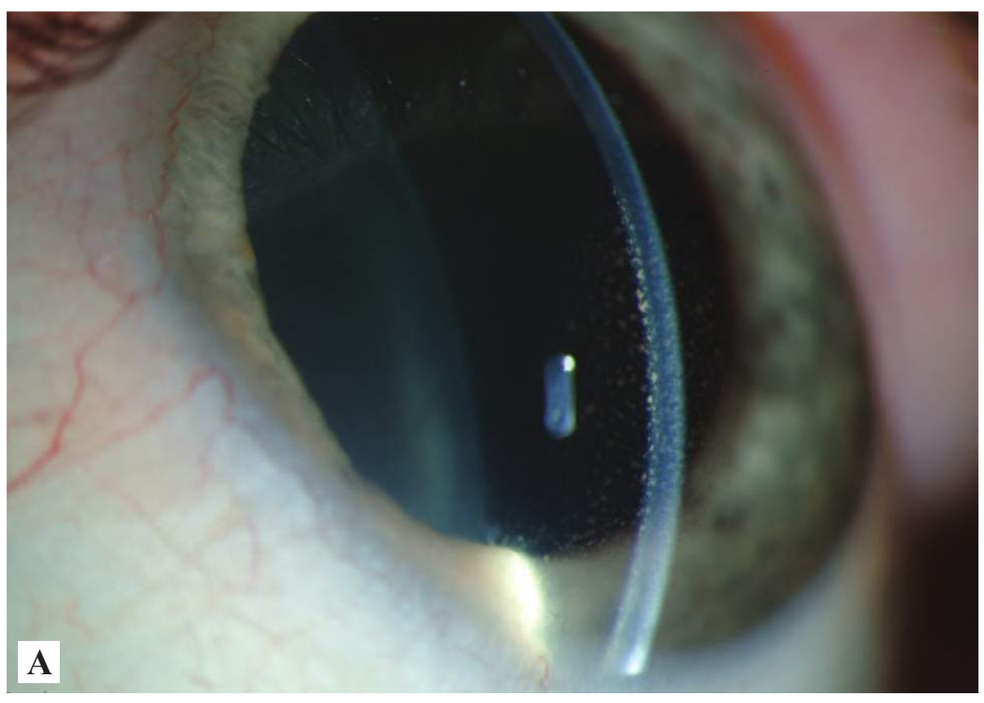

FIGURE 2.38 ■ **Keratic Precipitates.** Deposits of cells on the endothelial layer of the cornea are seen in these photographs with a slit-lamp beam (**A**), and under diffuse light (**B**). (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

SYMPATHETIC OPHTHALMIA

Clinical Summary

This rare disorder presents as an insidious or acute anterior uveitis of both eyes (exciting and sympathizing eye) after recent or remote trauma to the exciting eye. The initial injury is usually from prior penetrating eye trauma with delayed wound closure or, less commonly, from prior eye surgery.

The disease is thought to be an autoimmune inflammatory response against choroidal melanocytes released due to trauma. It may develop rapidly or insidiously, presenting from days to years after the inciting traumatic event. Most cases develop within 1 year.

Earliest symptoms include eye floaters and loss of accommodation. The disease may progress to severe uveitis with redness, pain, and photophobia in the sympathizing, noninjured eye.

Classically, there is a granulomatous anterior chamber reaction with mutton-fat keratic precipitates seen on the corneal endothelium.

Management and Disposition

Diagnosis is suspected clinically. Consider other causes of granulomatous uveitis such as sarcoidosis, tuberculosis, and syphilis. Consult ophthalmology for management, which may include topical steroids, evisceration, or enucleation of the eye with prior trauma.

Pearls

1. Consider sympathetic ophthalmia with unexplained uveitis and a history of penetrating eye trauma.
2. Solicit a history of eye trauma or prior eye surgery when evaluating eye pain, blurred vision, and redness.
3. Early intervention can be sight saving.

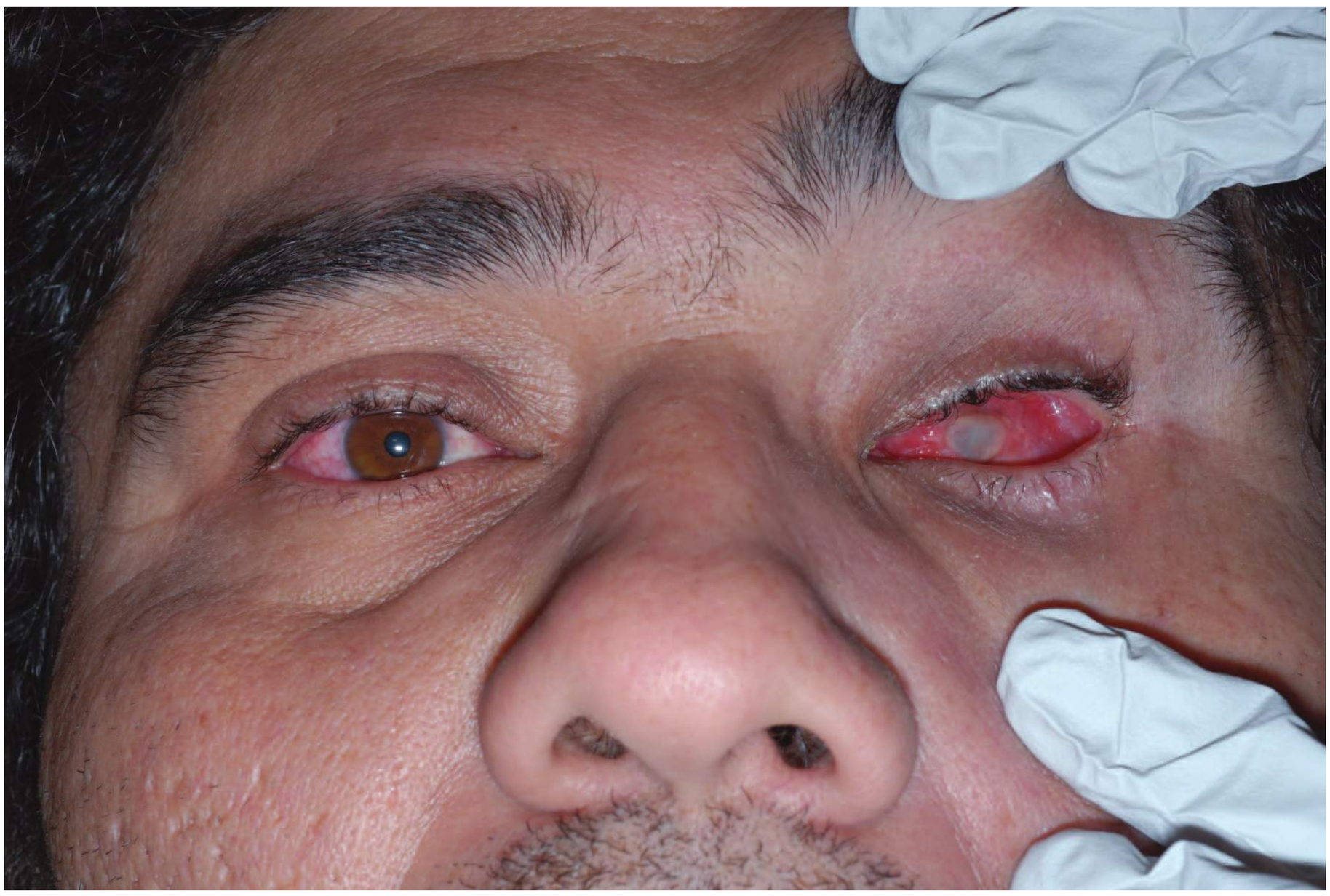

FIGURE 2.39 ■ **Sympathetic Ophthalmia.** This patient with prior ocular trauma (untreated globe rupture) OS presented with pain and redness OD, thought to be due to sympathetic ophthalmia. OS enucleation and treatment with steroids resolved condition. (Photo contributor: R. Jason Thurman, MD.)

ANISOCORIA

Clinical Summary

Anisocoria is a disparity of pupil size. To determine the abnormal pupil, compare pupil sizes in light and dark. It is accentuated in the paretic muscle. If the iris sphincter (or its innervation) is involved, the anisocoria will be increased in bright light. If the iris dilator muscle (or its innervation) is affected, it will be more pronounced in darkness. Up to 20% of normal individuals have physiologic anisocoria of 1 to 2 mm.

Other causes of anisocoria with an abnormally large pupil include mydriatic drops, contamination from a scopolamine patch, an Adie pupil, and ocular trauma with iris sphincter damage. A dilated pupil due to anticholinergic agents (eg, atropine) does not react to light, although a dilated pupil due to sympathomimetics still has some response.

An abnormally small pupil may be secondary to Horner syndrome, chronic Adie pupil (8 weeks or more after the event), iritis, and eye drops (pilocarpine). Miosis secondary to pupillary sphincter muscle spasm may be transiently observed after ocular trauma, followed by mydriasis.

Management and Disposition

Evaluation is dependent on clinical presentation. A patient with the acute onset of third-nerve palsy with associated headache or trauma should be evaluated as a neurosurgical emergency.

Pilocarpine may be helpful to differentiate the etiology of pupil dilation. With low concentrations (0.125%), an Adie pupil will constrict more than the unaffected eye secondary to denervation supersensitivity. With higher concentrations (1%), a pupil that fails to constrict is most likely secondary to topical anticholinergic mydriatics (scopolamine, atropine, or cyclopentolate). Unlike the mydriasis seen with intracranial pathology, pharmacologic mydriasis is not associated with pain, ptosis, or diplopia. Other pathology within the iris sphincter muscle that prevents constriction to 1% pilocarpine includes iris muscle trauma and synechiae.

Pearls

1. A patient with a dilated pupil, ptosis, and abnormal extraocular movements should be evaluated emergently for an aneurysm or expanding supratentorial mass with tentorial herniation.
2. With physiologic anisocoria, the pupil size disparity is the same in light and dark, and there are no other ocular findings.
3. A driver's license or ID badge may be helpful to document a preexisting anisocoria.
4. Some brands of eye make-up contain belladonna alkaloids which can cause mydriasis.
5. An Adie pupil initially is dilated, but may become miotic over time. It is a benign condition that affects young adults, women more often than men, and is associated with decreased reflexes.

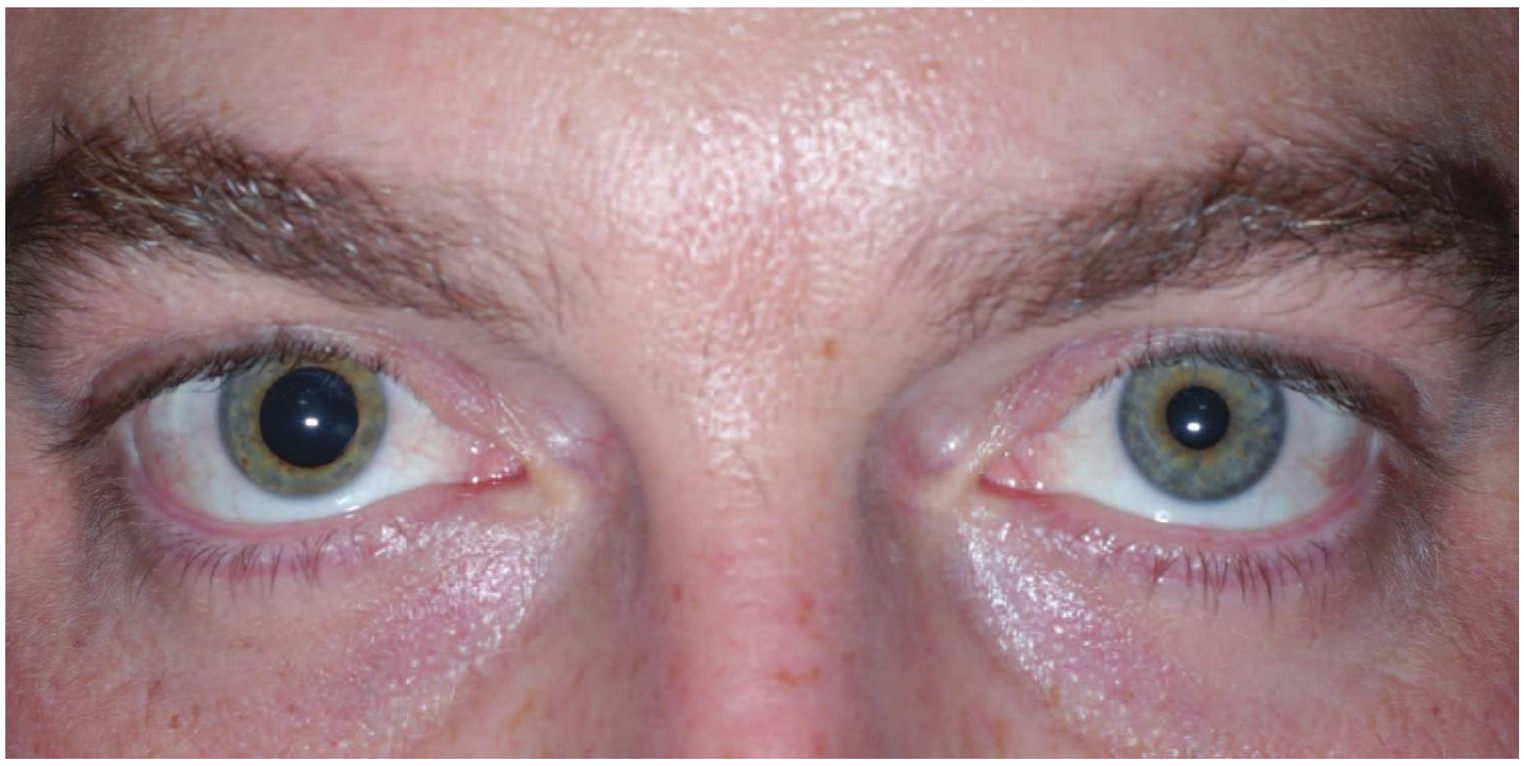

FIGURE 2.40 ■ **Posttraumatic Anisocoria.** Marked chronic anisocoria secondary to prior trauma as a child. (Photo contributor: R. Jason Thurman, MD.)

HERPES ZOSTER OPHTHALMICUS

Clinical Summary

Reactivation of endogenous latent varicella-zoster virus within the trigeminal ganglion with neuronal spread through the ophthalmic branch results in crops of grouped vesicles on the forehead and periocularly.

Patients typically present with periocular rash and an injected eye, along with a watery discharge. The most common corneal lesion is punctate epithelial keratitis, in which the cornea has a ground-glass appearance because of stromal edema. Pseudodendrites, also very common, form from mucous deposition, are usually peripheral, and stain moderate to poorly with fluorescein. These may be differentiated from the dendrites of herpes simplex in that the pseudodendrites lack the rounded terminal bulbs at the end of the branches, and are broader and more plaquelike. Anterior stromal infiltrates may be seen in the second or third week after the acute infection. Follicles (hyperplastic lymphoid tissue that appears as gray or white lobular elevations, particularly in the inferior cul-de-sac) and regional adenopathy may or may not be present. Iritis is seen in approximately 40% of patients.

Management and Disposition

Treat patients with epithelial defects with topical broad-spectrum antibiotics to prevent secondary infection. Initiate oral antivirals within 72 hours of onset, and treat for 7 to 10 days. Use cycloplegics if an iritis is present. Artificial tears or ointment may be helpful and narcotic analgesics may be required. Ophthalmologic consultation is indicated.

Pearls

1. Ocular complications may follow the rash by many months to years. These complications have a highly variable presentation that can mimic almost any ophthalmic condition.
2. Recurrence is more common in the immunocompromised host.
3. Perform a careful eye exam with corneal staining. Nearly two-thirds of patients will develop ocular lesions.
4. Corneal hypesthesia and the appearance of dendrites with fluorescein staining are seen in both herpes zoster ophthalmicus and herpes simplex keratitis.
5. Patients with skin lesions on the tip of the nose (Hutchinson sign) are at high risk for ocular involvement. However, the eye may be involved without a nasal lesion.

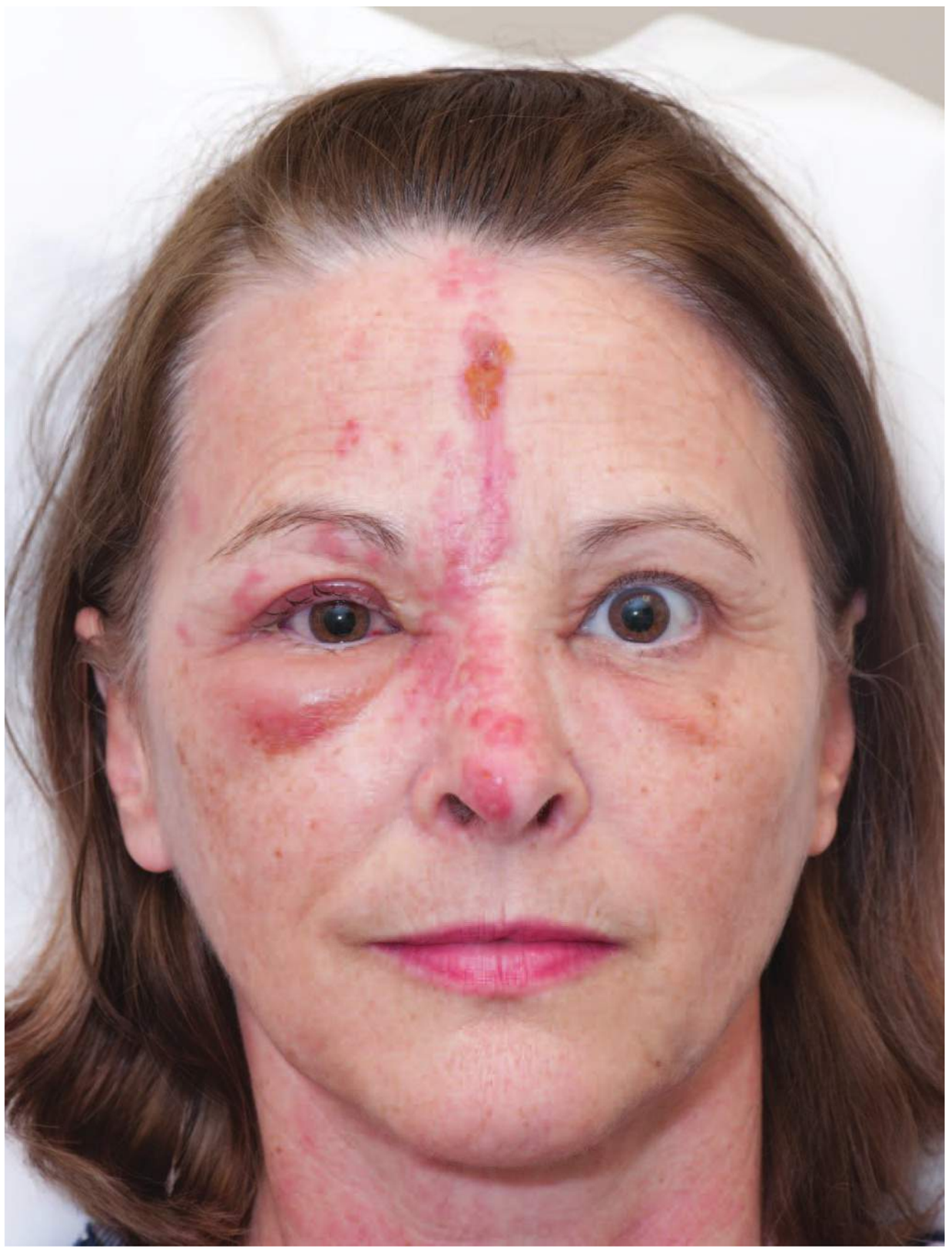

FIGURE 2.41 ■ **Herpes Zoster Ophthalmicus.** A vesicular rash in the distribution of the ophthalmic division (V1) of the trigeminal nerve is seen. The presence of the lesion near the tip of the nose (Hutchinson sign) increases the risk of ocular involvement. (Photo contributor: Lawrence B. Stack, MD.)

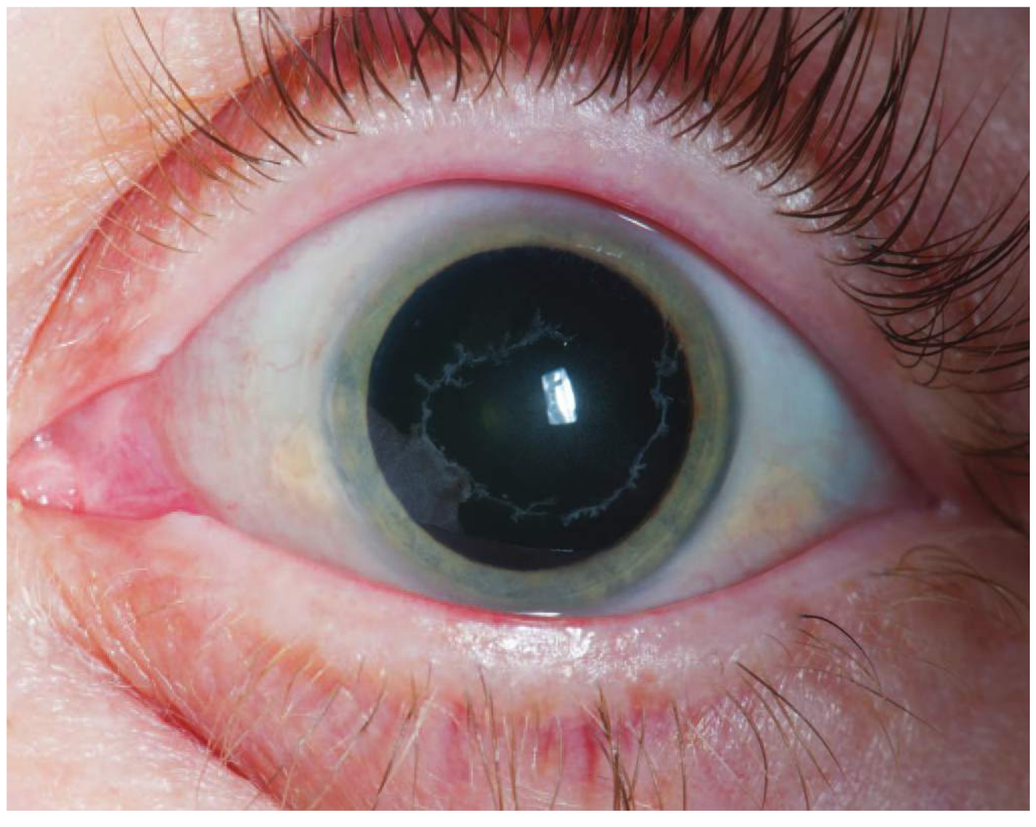

FIGURE 2.42 ■ **Herpes Zoster Ophthalmicus.** A large circular dendrite is seen in this patient with ocular involvement from HZV. (Photo contributor: Alexandra Bingnear, RN.)

OCULAR HERPES SIMPLEX

Clinical Summary

Ocular herpetic disease may be neonatal, primary, or recurrent. Neonatal disease occurs secondary to passage through an infected birth canal, and is usually HSV type 2. Primary ocular herpes may present as a blepharitis (grouped eyelid vesicles on an erythematous base), conjunctivitis, or keratoconjunctivitis. Patients with keratoconjunctivitis commonly note pain, irritation, foreign body sensation, redness, photophobia, tearing, and occasionally decreased visual acuity. Follicles and preauricular adenopathy may be present. Initially, the keratitis is diffuse and punctate, but after 24 hours, fluorescein demonstrates either serpiginous ulcers or multiple diffuse epithelial defects. True dendritic ulcers are rarely seen in primary disease.

Most ocular herpetic infections are manifestations of recurrent disease rather than a primary ocular infection. These may be triggered by ultraviolet laser treatment, topical ocular medications (β-blockers, prostaglandins), and immunosuppression (especially ophthalmic topical glucocorticoids). Recurrent disease most commonly presents as keratoconjunctivitis with a watery discharge, conjunctival injection, irritation, blurred vision, and preauricular lymph node involvement. Corneal involvement initially is punctate, but evolves into a dendritic keratitis. The linear branches classically end in bead-like extensions called terminal bulbs. Fluorescein dye demonstrates primarily the corneal defect; the terminal bulbs are best seen with rose stain. In addition to the dendritic pattern, fluorescein stain may instead take on a geographic or ameboid shape, secondary to widening of the dendrite. Most patients (80%) with herpes simplex keratitis have decreased or absent corneal sensation in the area of the dendrite or geographic ulceration. Deeper corneal stromal inflammation may also occur (disciform keratitis). Recurrent disease can also present with iritis or with blepharitis, with vesicles grouped in focal clusters.

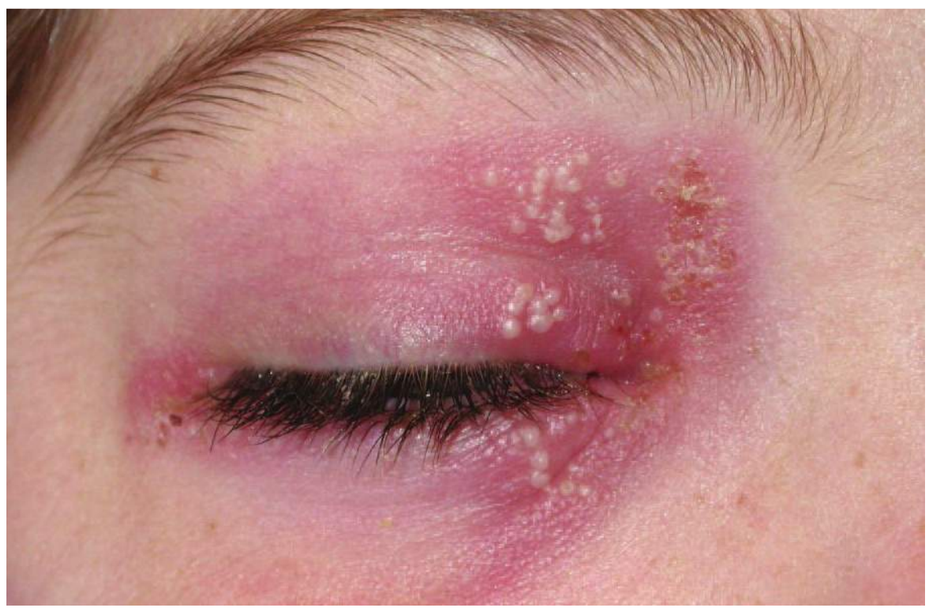

FIGURE 2.43 ■ **Ocular Herpes Simplex.** This 18-year-old has a history of ocular herpes simplex since childhood. Grouped vesicles on an erythematous base with periorbital erythema are seen. Honey-colored crusts suggest secondary impetigo. (Photo contributor: Lawrence B. Stack, MD.)

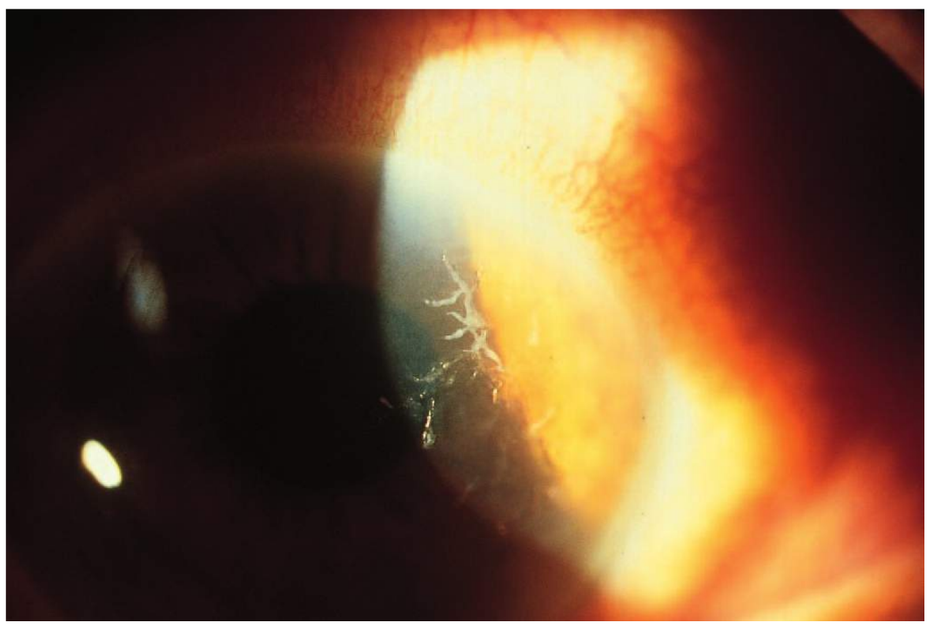

FIGURE 2.44 ■ **Herpes Simplex Keratitis.** A slit-lamp view of unstained dendritic lesions. (Photo contributor: Lawrence B. Stack, MD.)

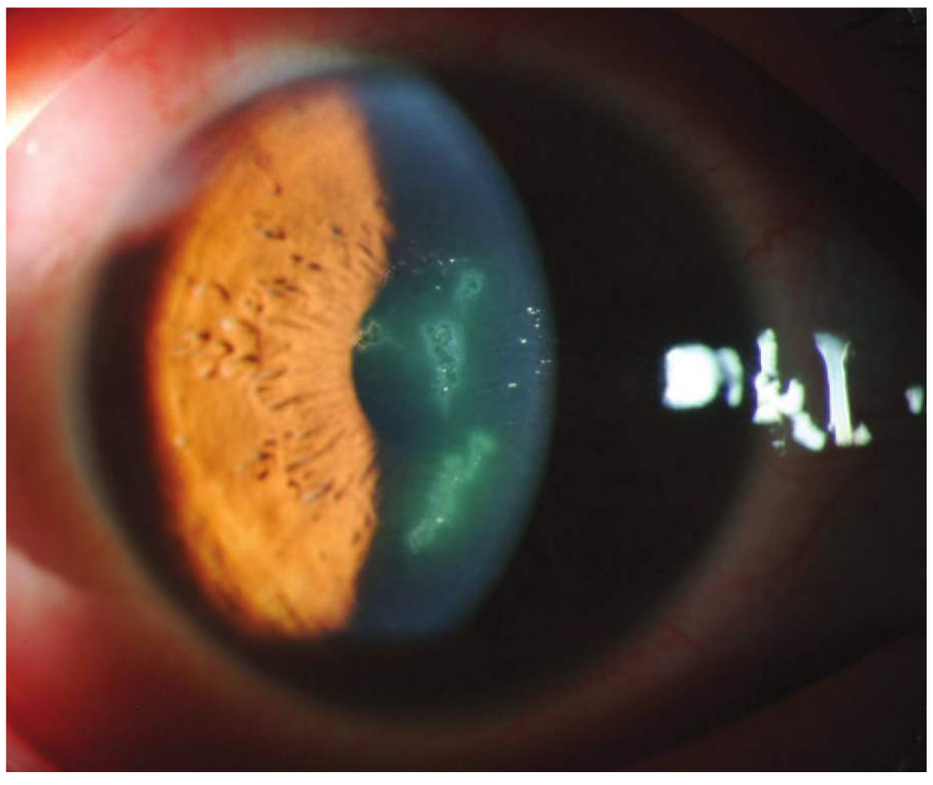

FIGURE 2.45 ■ **Herpes Simplex Keratitis.** A slit-lamp view of dendritic lesions under white light. (Used with permission from The University of Iowa and EyeRounds.org.)

Management and Disposition

There is a high association in neonatal ocular herpes infections between ocular HSV disease and serious systemic or neurologic infection, and an emergency pediatric or infectious disease consult is necessary. IV acyclovir is indicated, and the ocular disease itself may be treated with topical antivirals (idoxuridine, vidarabine, trifluorothymidine). Other sexually transmitted diseases such as chlamydia or gonorrhea should be explored.

Treatment of those with primary ocular herpes (beyond the neonatal period) presenting as blepharitis or periocular dermatitis consists of good local hygiene and a prophylactic topical antiviral such as trifluorothymidine or idoxuridine ointment. Patients with corneal involvement should additionally receive topical antibiotics to prevent secondary bacterial infection.

In those with recurrent disease, topical antivirals (trifluridine, ganciclovir, acyclovir, vidarabine) are effective, as is oral acyclovir. If the cornea is involved, trifluorothymidine and topical antibiotics are added. Ophthalmology consultation is required in patients with ocular HSV disease. Episodes of recurrent stromal disease may be limited by the long-term use of low-dose oral antivirals.

Pearls

1. Most ocular HSV infections are manifestations of recurrent disease.
2. Corneal disease is the most prevalent form of ocular HSV disease.
3. Oral antivirals are now frequently used for keratitis because of their convenience, although topical optical antivirals are equally effective.
4. HSV dendrites, when stained with fluorescein, appear as branching lesions with terminal bulbs.
5. Corneal hypesthesia may be easily overlooked in the initial evaluation of a red eye. If a topical anesthetic has been given, a reexamination 1 hour later is helpful for evaluating hypesthesia.

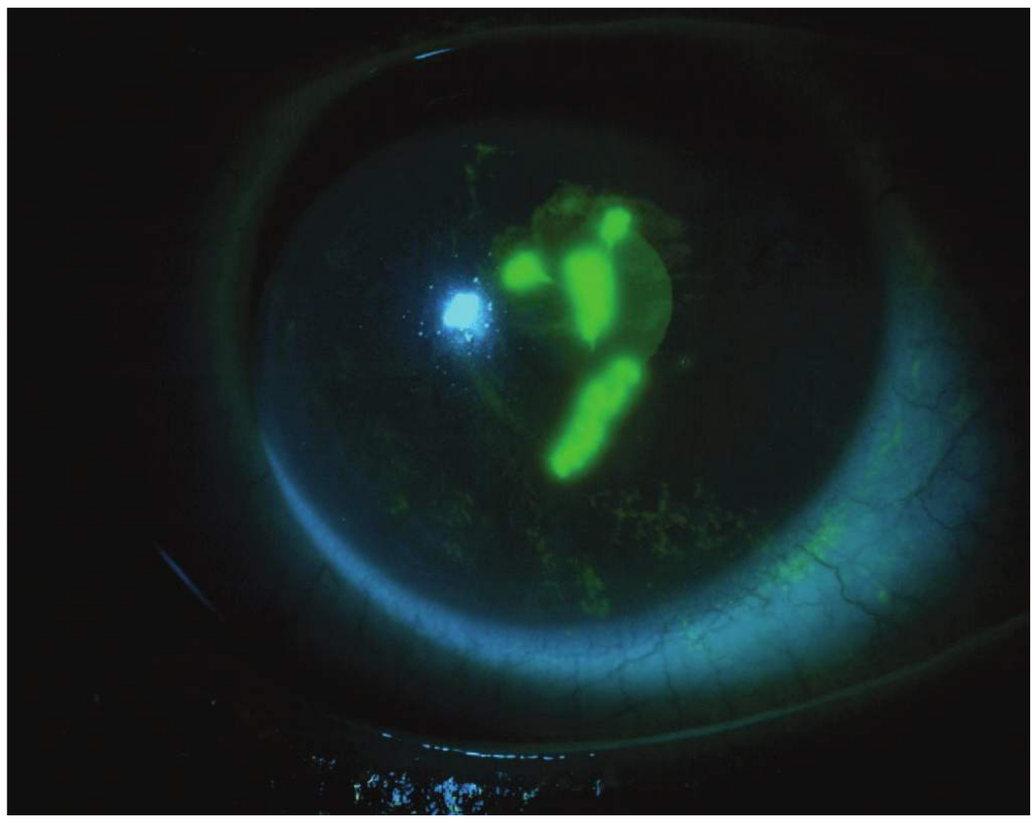

FIGURE 2.46 ■ **Herpes Simplex Keratitis.** Epithelial dendrites seen in Fig. 2.45 after fluorescein staining. (Used with permission from The University of Iowa and EyeRounds.org.)

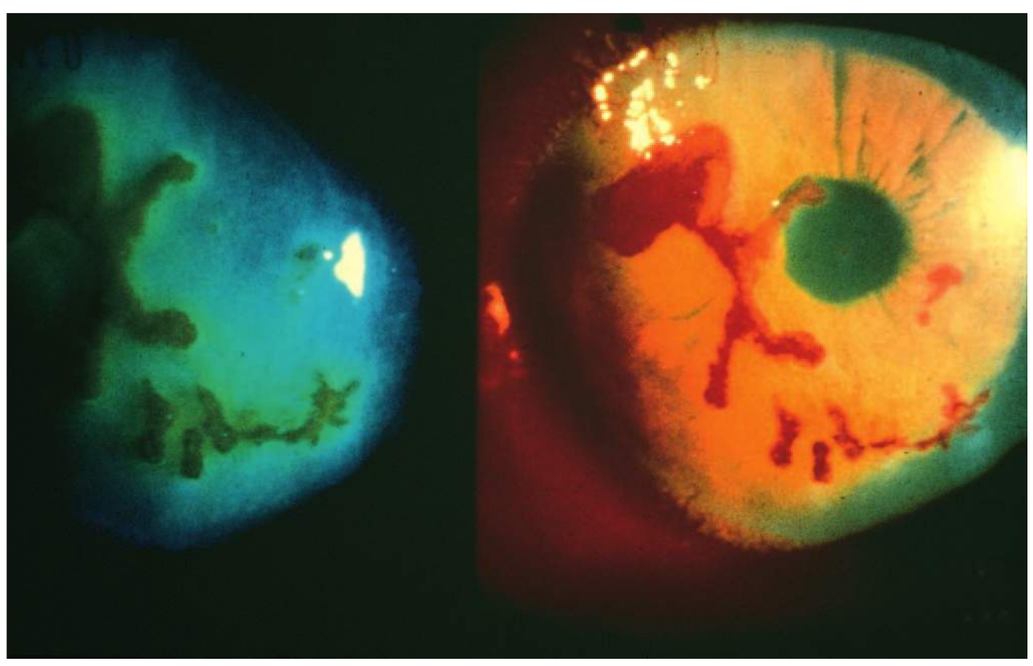

FIGURE 2.47 ■ **Herpes Simplex Keratitis**. Fluorescein (left) and rose bengal (right) stains demonstrate characteristic dendritic patterns. Whereas fluorescein staining is used to detect epithelial defects, rose bengal staining additionally demonstrates degenerating or dead epithelial cells and is particularly good for demonstrating the club-shaped terminal bulbs at the end of each branch. (Used with permission from the American Academy of Ophthalmology. External Disease and Cornea: A Multimedia Collection. San Francisco; 1994.)

Clinical Summary

A corneal ulcer is an inflammatory and ulcerative keratitis. Common infectious etiologies include bacteria (Staphylococcus, Streptococcus, Pseudomonas) and viruses (herpes simplex, adenovirus). Bacterial corneal ulcers are commonly associated with extended-wear contact lenses. Rare causes of corneal ulcers include fungal infections and Acanthamoeba, a ubiquitous protozoan associated with contaminated contact lens solutions. Fungal infections may also arise from trauma involving vegetable matter such as a tree branch. Acanthamoeba infections may also occur from swimming in lakes, especially while wearing contact lenses.

Patients present with pain, photophobia, decreased vision, discharge, and a foreign body sensation. Ocular findings include a corneal infiltrate, typically a round white spot, with conjunctival hyperemia, meiosis, and chemosis. Slit-lamp biomicroscopy may demonstrate an epithelial defect with fluorescein uptake. Anterior chamber findings can include cells and flare, keratic precipitates, and a hypopyon.

Management and Disposition

Corneal ulcers are an ophthalmologic emergency requiring emergent ophthalmology consultation. Stains and cultures should be obtained as expeditiously as possible. Intensive topical treatment using fortified antibiotics is the most effective treatment route, initially given every 30 to 60 minutes. For mild cases, a single fluoroquinolone agent may suffice. For more severe cases, dual therapy using a cephalosporin or vancomycin combined with an aminoglycoside is recommended. Clinical improvement is usually noted after 2 to 3 days. Systemic antibiotics are used in cases where the sclera is involved (Pseudomonas) or if there is a high risk of concurrent systemic disease (Neisseria, Haemophilus). Cycloplegics are recommended if there is an accompanying iritis. Steroids and eye patching are contraindicated. A contact lens wearer must discontinue contact lens wear.

Pearls

1. A corneal ulcer is an ophthalmologic emergency.
2. Extended-wear contact lens use is a risk factor for corneal ulcer.
3. Pseudomonas aeruginosa, associated with thick yellow-green or blue-green mucopurulent tenacious exudate, is capable of destroying the cornea within 6 to 12 hours.
4. Acanthamoeba should be suspected in contact lens wearers with contaminated lens solutions or who swim wearing their contact lens. Classically, these patients have pain out of proportion to their clinical findings.
5. Infectious ulcers tend to develop centrally, away from the vascular supply and immune system of the limbus.

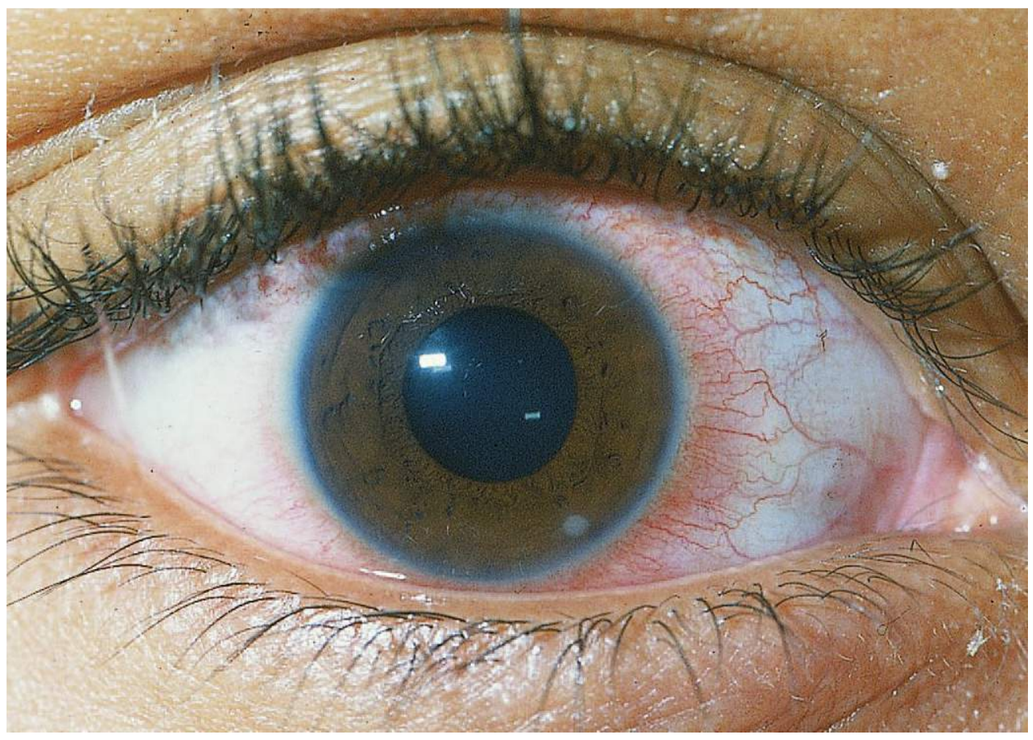

FIGURE 2.48 ■ **Corneal Ulcer.** An elliptical ulcer at 5-o'clock position near the limbus is seen. This location is atypical for a bacterial ulcer. The patient presented with painful red eyes and normal uncorrected vision, but wore cosmetic soft contact lenses. Bilateral corneal ulcers were diagnosed, which cleared after treatment with topical ciprofloxacin. Note that the ciliary flush seen in the nasal portion of the limbus is not to be mistaken for conjunctivitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

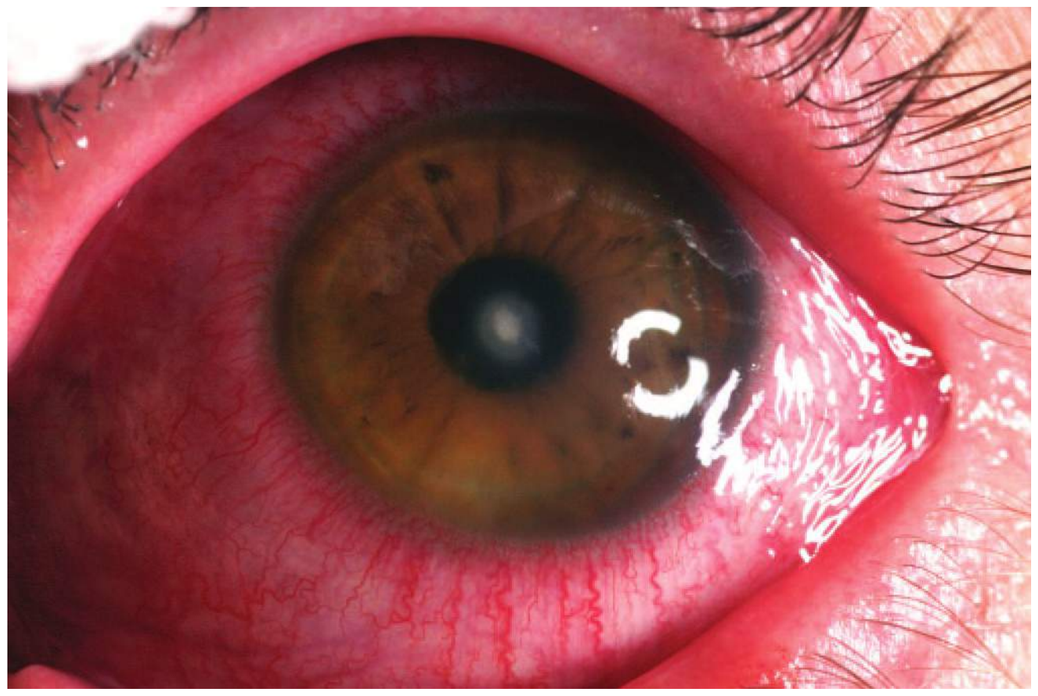

FIGURE 2.49 ■ **Corneal Ulcer.** A faint white circular corneal infiltrate is seen in the central visual axis. (Photo contributor: Kevin J. Knoop, MD, MS.)

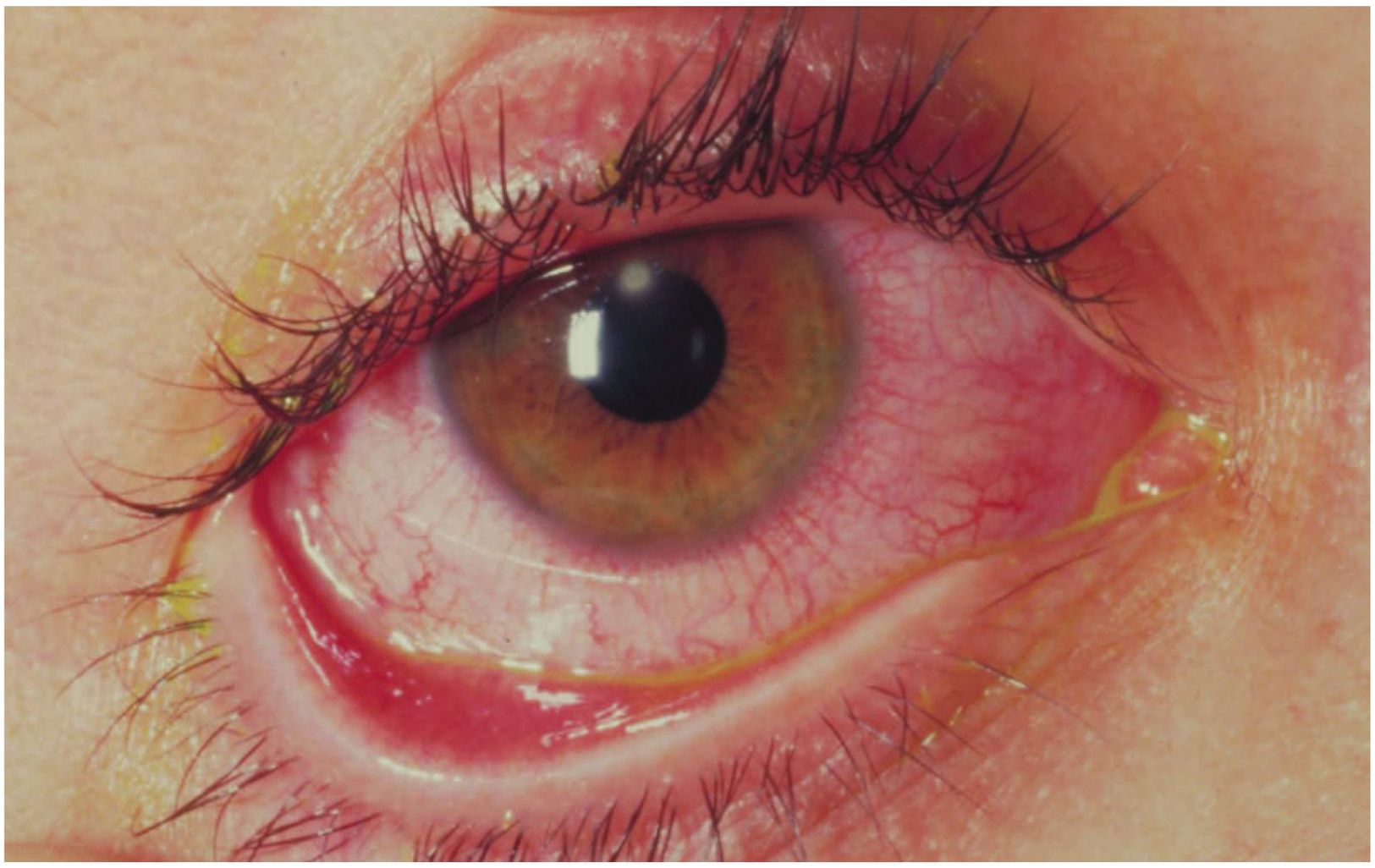

FIGURE 2.50 ■ **Corneal Ulcer**. A small circular corneal infiltrate is seen adjacent to the white flash photography reflection. Diffuse conjunctival hyperemia with a nasal ciliary flush is seen. (Photo contributor: Lawrence B. Stack, MD.)

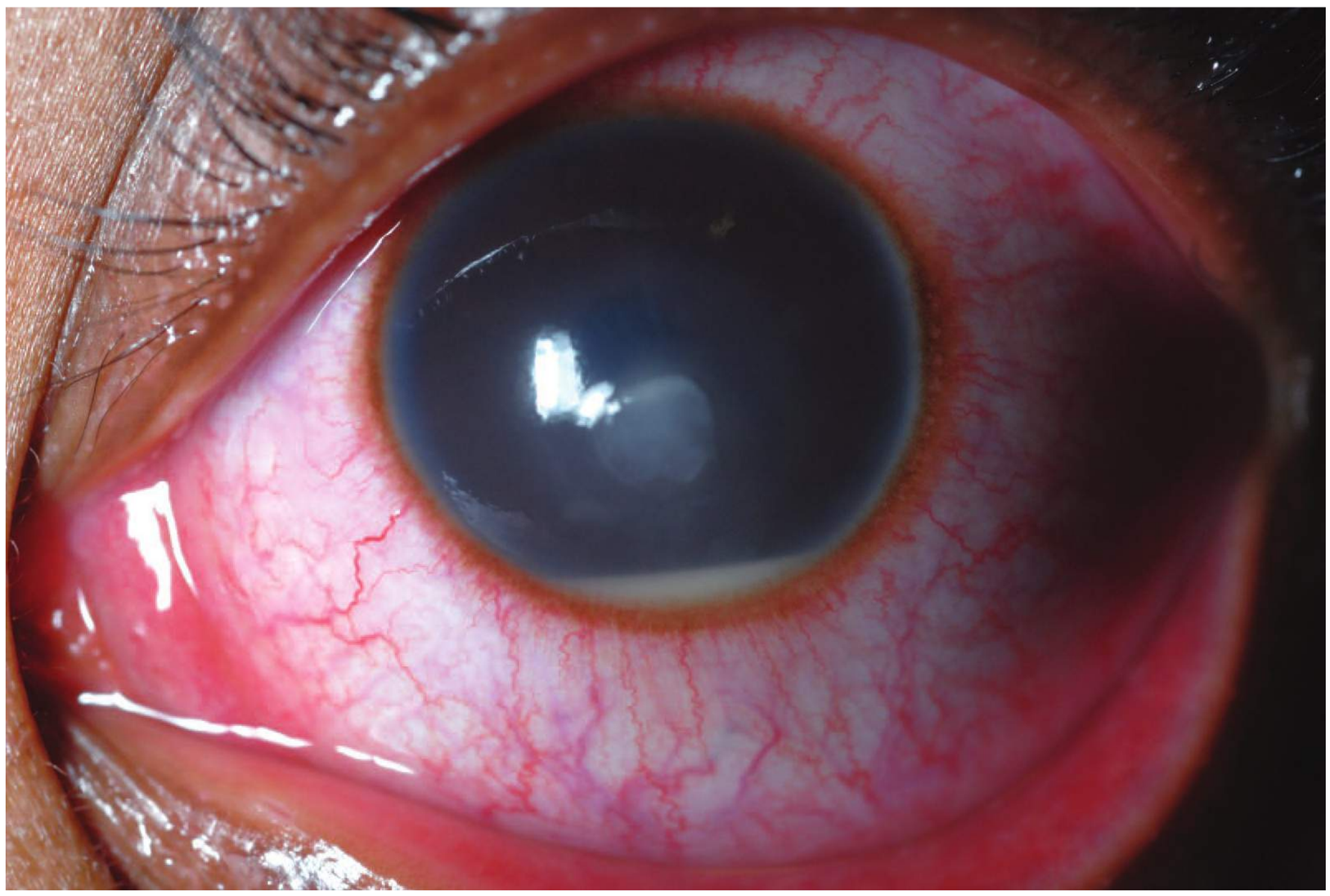

FIGURE 2.51 ■ **Corneal Ulcer with Hypopyon.** A hypopyon has developed from a corneal ulcer seen near the visual axis. The conjunctiva is inflamed and a ciliary flush is present. (Photo contributor: R. Jason Thurman, MD.)

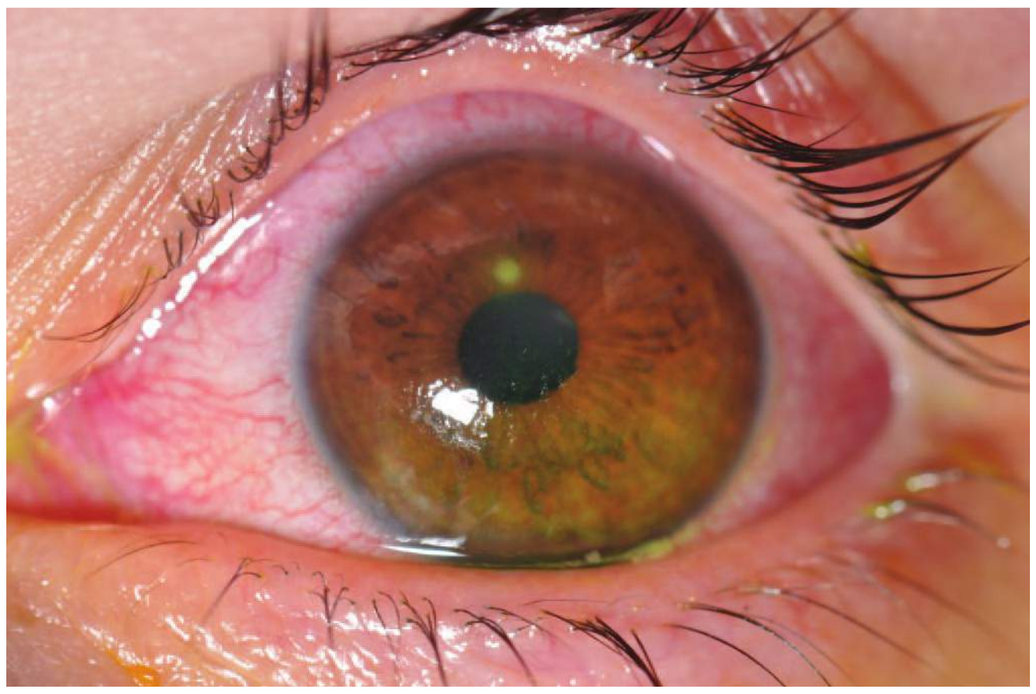

FIGURE 2.52 ■ **Corneal Ulcer.** Diminished visual acuity and pain due to the corneal ulcer just superior to the visual axis contributed to his MVC and airbag deployment, causing a large corneal abrasion of the inferior third of the cornea. (Photo contributor: Lawrence B. Stack, MD.)

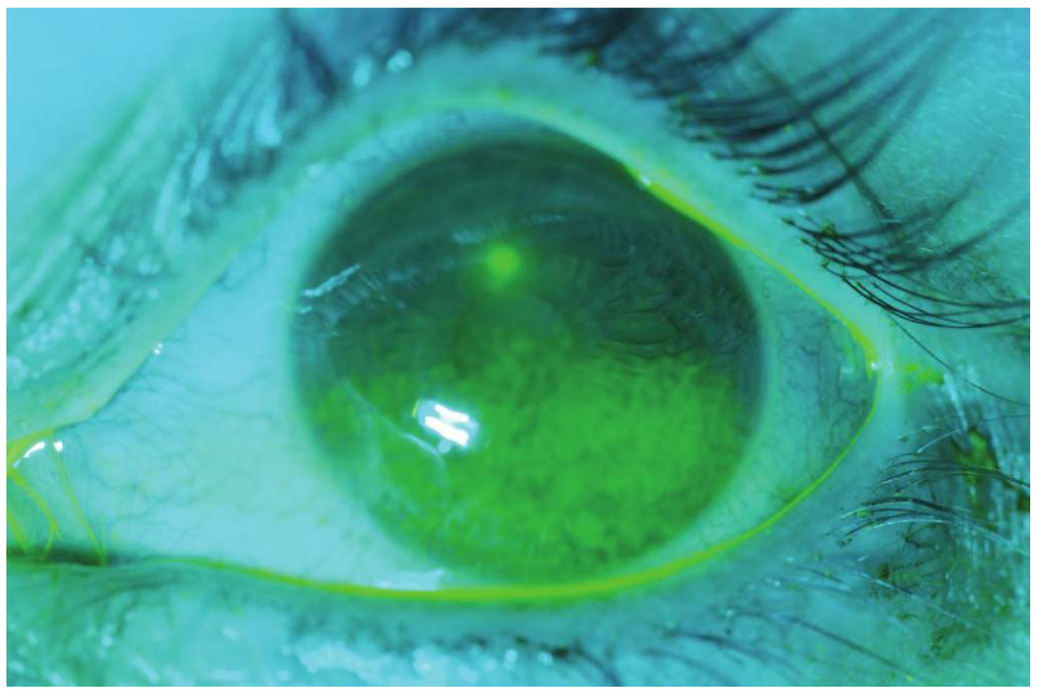

FIGURE 2.53 ■ **Corneal Ulcer.** Fluorescein stain uptake of the corneal ulcer just above the visual axis and the corneal abrasion of the inferior third of the corneal surface due to airbag deployment. (Photo contributor: Lawrence B. Stack, MD.)

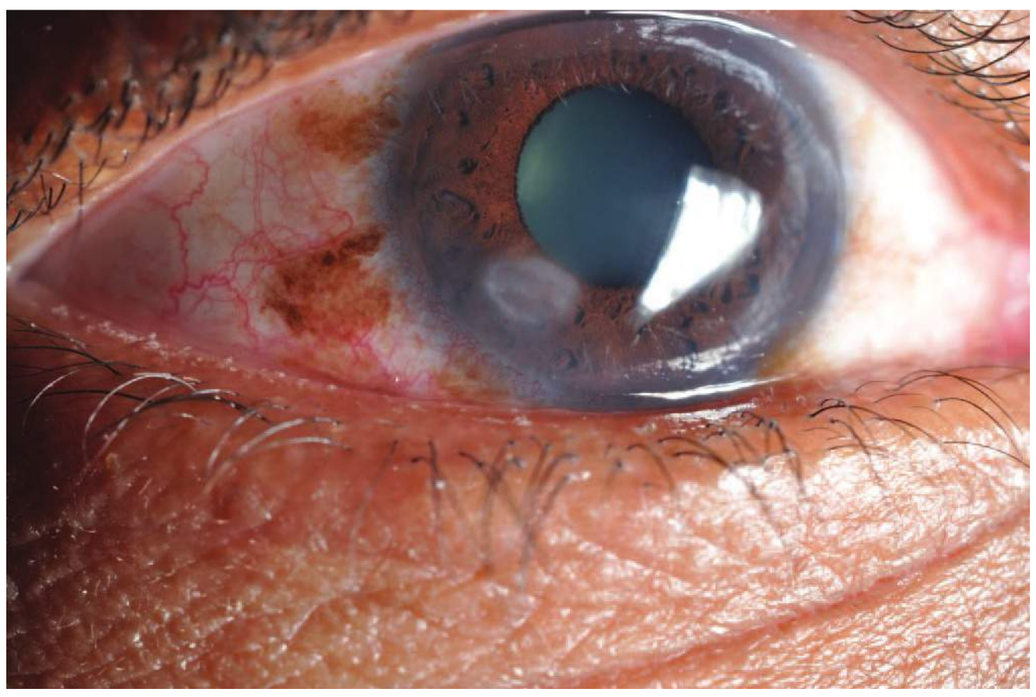

FIGURE 2.54 ■ **Corneal Ulcer**. A large oval opaque corneal ulcer is seen at 8-o'clock position toward the limbus in a contact-lens wearing patient. (Photo contributor: Lawrence B. Stack, MD.)

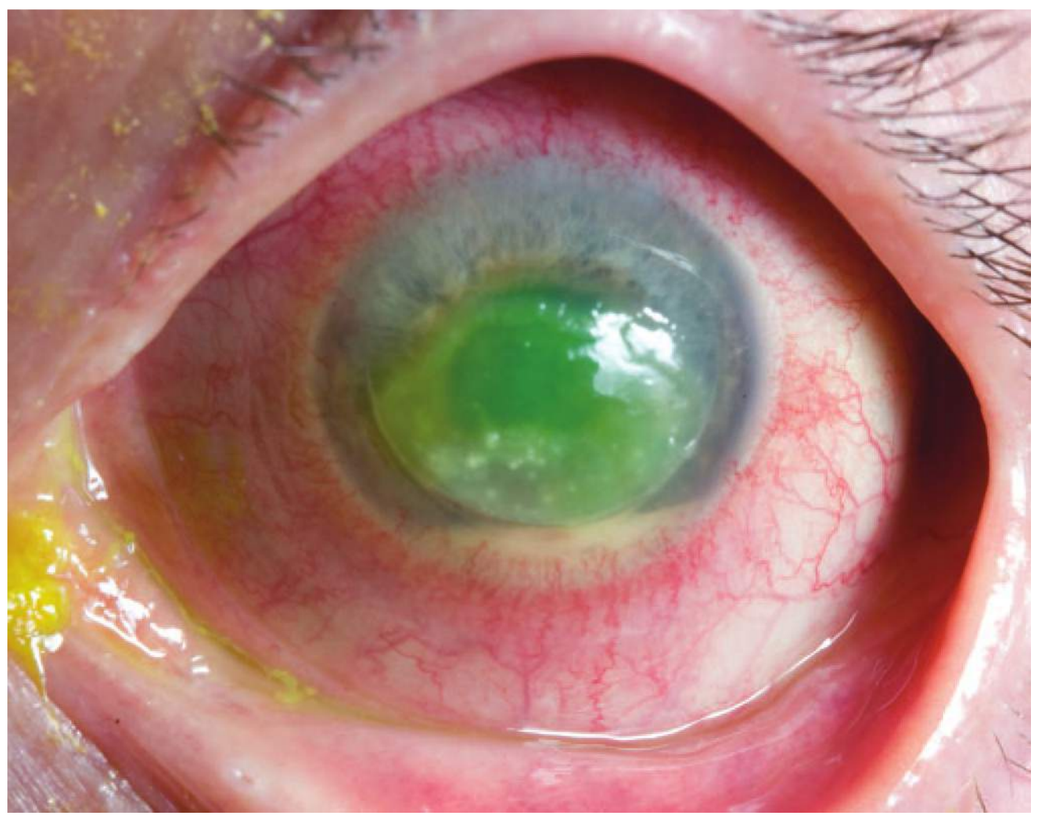

FIGURE 2.55 ■ **Corneal Ulcer with Hypopyon**. A middle-aged female accidentally splashed dishwashing detergent in her eye one week prior to ED visit. A large inferior corneal ulcer, with corneal edema and hypopyon, is seen. (Photo contributor: Eugene Eiland, MD.)

AFFERENT PUPILLARY DEFECT (MARCUS GUNN PUPIL)

Clinical Summary

Pupil size is controlled in the midbrain, from which efferent nerves travel to both pupils resulting in symmetric pupils, even with a unilateral light stimulus. The perception of the light stimulus, however, may be decreased by disease within anterior visual structures such as the retina, optic nerve, chiasm, optic tract, and midbrain pathways. With diminished afferent stimulation, less light is "perceived," and pupil contraction diminishes (ie, dilates) as a result. In this situation, the affected side is said to demonstrate an afferent pupillary defect (APD), and the pupil is called a Marcus Gunn pupil.

A Marcus Gunn pupil is best appreciated by the swinging flashlight test, which discloses differences in afferent stimuli between the two eyes. A flashlight is directed onto one pupil and then the other. The normal response is a prompt constriction. (Generally, this is followed by a slight "release" dilation.) During the brief interval required to move the light from one to the other eye, both pupils begin to dilate. Thus, under normal circumstances, when the light reaches the second eye, reflex constriction again occurs. With a diseased eye or pathway, the diseased side perceives less light and the midbrain therefore sets the pupil size to be larger, and as a result the pupils dilate (or continue to dilate following the "release" dilation).

Although sensitive, the swinging flashlight test is not specific, since the pathology may be anywhere in the visual pathway from the retina to the midbrain. The test cannot distinguish between unilateral afferent disease and asymmetric bilateral disease. Typically, a positive finding indicates optic nerve disease such as ischemic optic neuropathy, optic neuritis (including that seen with multiple sclerosis), retrobulbar optic neuritis, and glaucoma.

Management and Disposition

The finding of a Marcus Gunn pupil is nonspecific. A fundoscopic exam may show occlusion of the central retinal vein or central retinal artery, or a dense hemorrhage in the vitreous. Otherwise, a neurologic evaluation (history, physical examination, and CT scan) is important to assess for treatable conditions. In the absence of gross ocular or neurologic disease, a clinically stable patient may be discharged from the ED with ophthalmology follow-up.

Pearls

1. An APD does not cause anisocoria because any change in light input results in a bilaterally symmetric output from the midbrain.
2. In the swinging flashlight test, both pupils will dilate when the flashlight moves from the normal pupil to the Marcus Gunn pupil, since pupil size is centrally mediated in the midbrain.
3. Bilaterally symmetric disease produces no APD, since the basis for the swinging flashlight test is asymmetry of disease.
4. A dense vitreous hemorrhage is capable of producing a mild defect. Lesser ocular pathology such as branch retinal vein or artery occlusion or a cataract is unlikely to produce a clinically detectable APD.
5. Dim room illumination may be helpful when performing the swinging flashlight test. The patient should focus on an object 15 feet away to avoid the pupil constriction normally seen with accommodation.

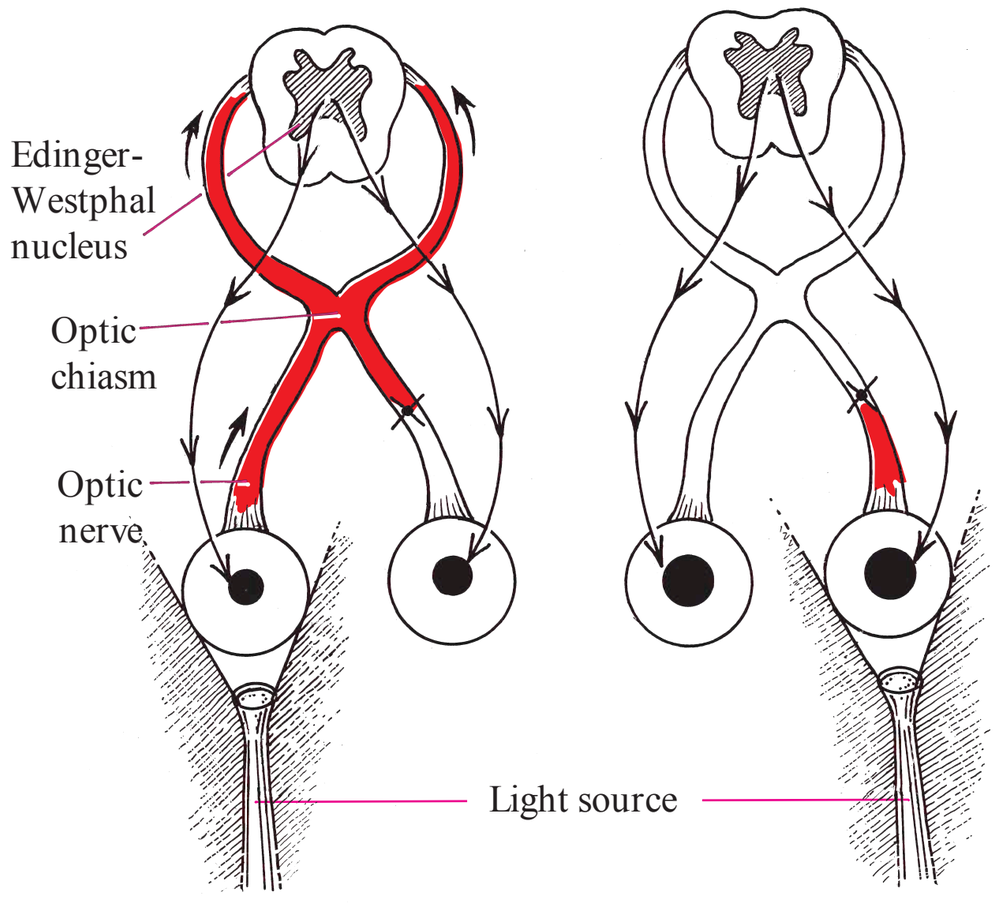

FIGURE 2.56 ■ **Afferent Pupillary Defect.** Schematic representation of an afferent pupillary defect (APD) due to neurologic lesion in the anterior visual pathway.

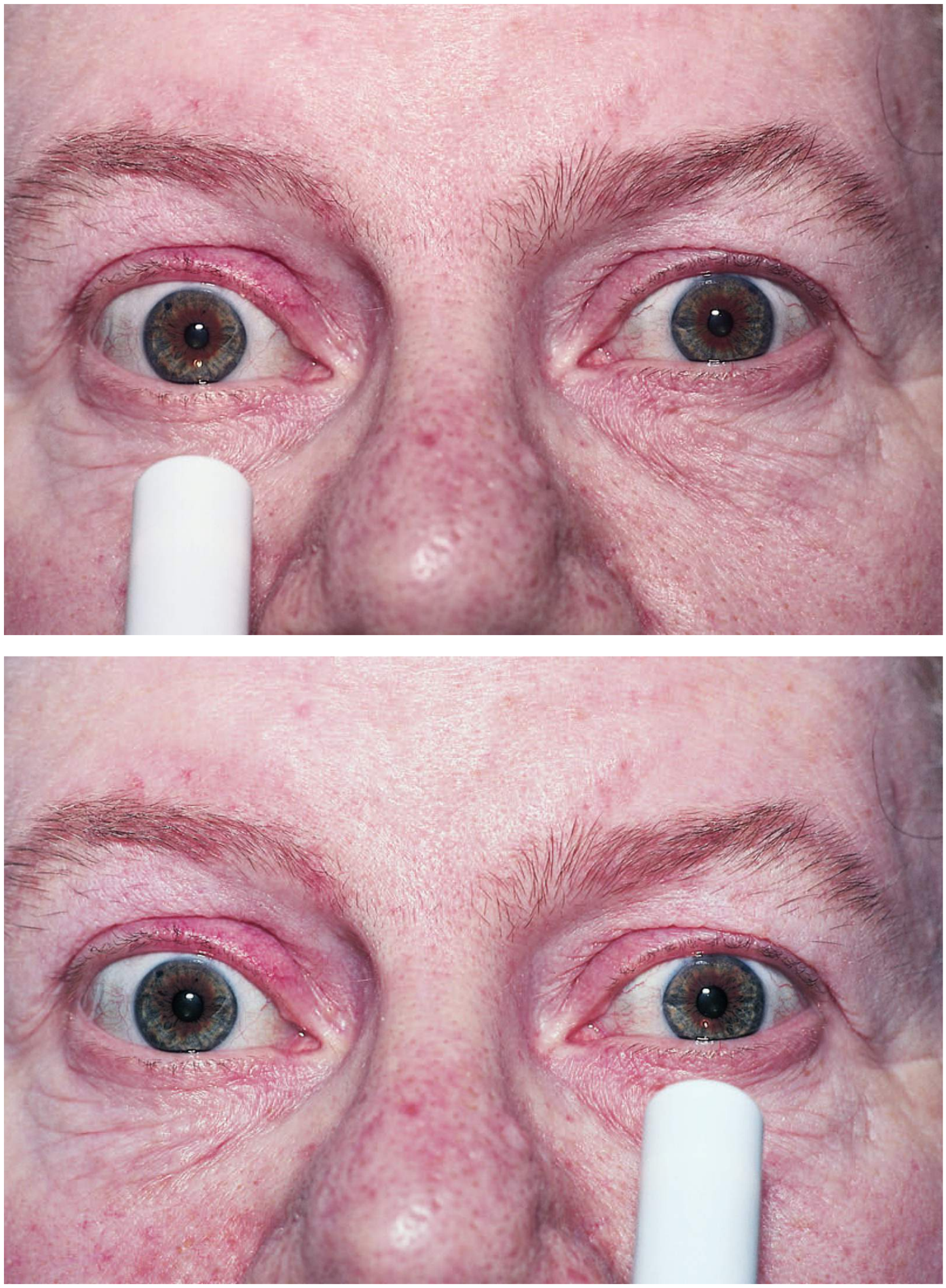

FIGURE 2.57 ■ **Marcus Gunn Pupil**. These photographs demonstrate an afferent pupillary defect of the left pupil. When the light shines in the affected left eye, the pupils are less constricted than when the light shines in the right eye. Thus, when the light is swung from the right pupil to the left, the pupils appear to dilate. The anisocoria here is subtle. (Photo contributor: Frank Birinyi, MD.)

Clinical Summary

Horner syndrome (miosis, ptosis, and anhidrosis) is secondary to loss of ocular sympathetic innervation. Ptosis is less than 2 mm, the result of paralysis of Müller muscle, innervated by the sympathetic pathway. Anhidrosis is often not apparent to patients or clinicians. A pupillary finding specific in Horner syndrome is dilation lag. Because the dilator muscle is weak, the pupil dilates more slowly than the normal pupil.

This loss of ocular sympathetic innervation can be produced by a lesion anywhere along a three-neuron sympathetic pathway, from the hypothalamus down through the brain stem to the cervical cord, in the apex of the chest, along the carotid sheath, and in the cavernous sinus or orbit. Isolated Horner syndrome presenting with head or neck pain suggests an internal carotid artery dissection.

Management and Disposition

Associated signs and symptoms help direct the ED workup. A patient with cranial nerve abnormalities requires CT or MRI imaging and admission. In the setting of cervical spine trauma, neck immobilization and appropriate imaging studies are instituted. Consider carotid artery dissection in neck pain without trauma.

Pearls

1. Classic signs of a Horner syndrome include meiosis and mild ptosis. Anhidrosis is less readily appreciated, and occurs with first- or second-order lesions only.
2. Miosis and dilation lag are accentuated in a darkened room.
3. A Pancoast tumor usually is caused by bronchogenic carcinoma and may present, as its presenting sign, as a Horner syndrome.
4. Cluster headaches, with autonomic sympathetic system dysfunction, are capable of producing an ipsilateral Horner syndrome.
5. Ipsilateral extraocular paresis, especially a sixth-nerve palsy, without other brainstem signs localizes pathology to within the cavernous sinus.

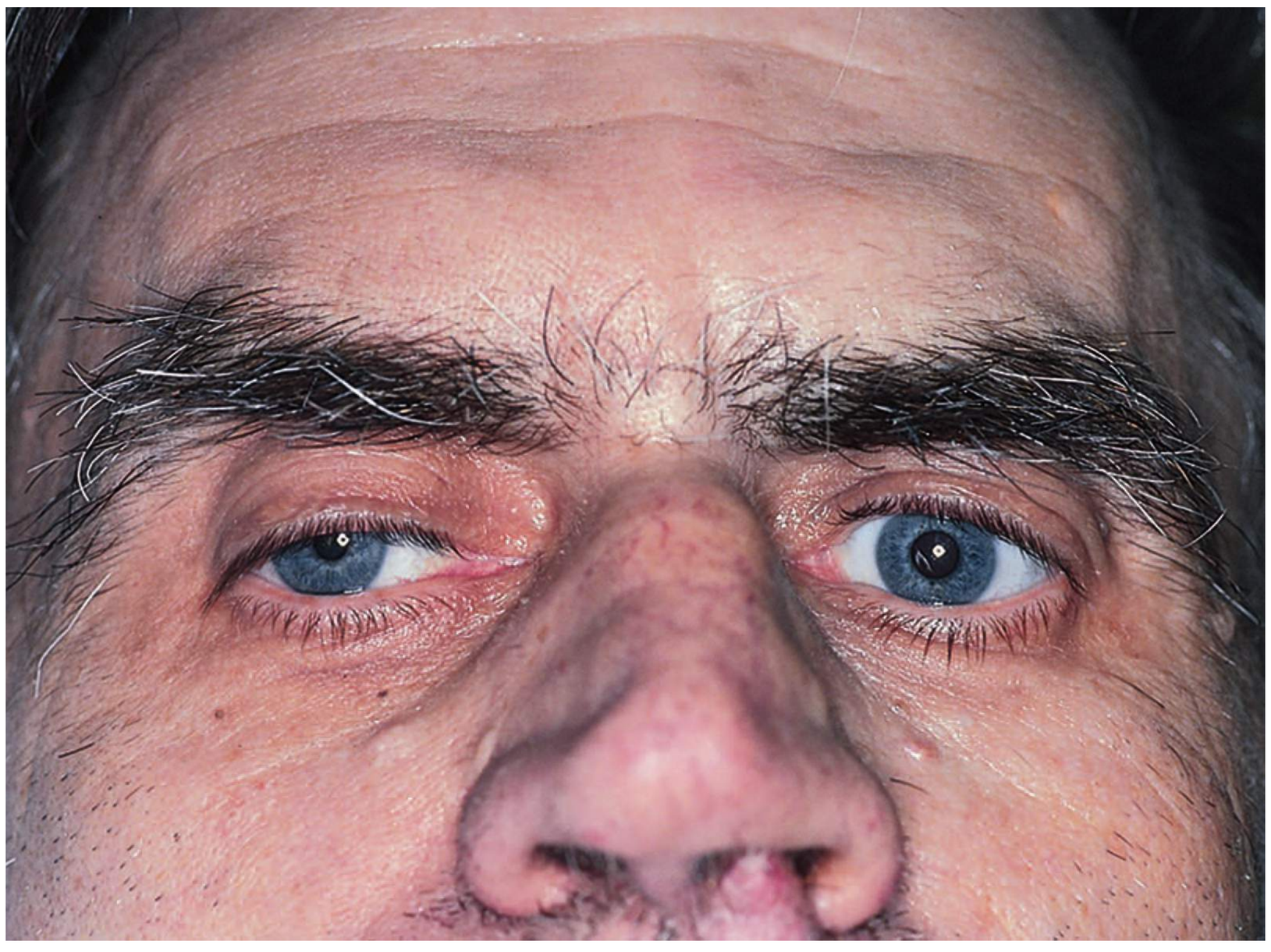

FIGURE 2.58 ■ **Horner Syndrome.** Unilateral meiosis and ptosis are seen in this patient with Horner syndrome and sarcoma metastatic to the spine. (Photo contributor: Frank Birinyi, MD.)

THIRD-NERVE PALSY

Clinical Summary

The third cranial nerve innervates all the extraocular muscles except the lateral rectus and superior oblique (LR6, SO4). It also controls the levator palpebrae muscle and supplies parasympathetics to the pupillary constrictor and ciliary muscles. Therefore, the clinical examination is notable for a dilated and unreactive pupil, limited extraocular movements, and ptosis. The eye rests in a position of abduction because of unopposed action of the lateral rectus.

Third-nerve dysfunction can result from lesions anywhere along its path from the oculomotor nucleus in the midbrain, within the subarachnoid space, traversing the cavernous sinus, and terminating in the extraocular muscles within the orbit. Contralateral hemiparesis suggests brainstem involvement (Weber syndrome). Pathology within the subarachnoid space causing a third-nerve palsy includes compression of the nerve by a posterior communicating artery aneurysm, uncal herniation, or compressive neoplasm.

Pathology within the cavernous sinus causing a third-nerve palsy includes carotid artery aneurysm, cavernous sinus thrombosis, and carotid-cavernous fistula. Third-nerve lesions here are often accompanied by lesions involving the fourth, fifth (ophthalmic branch), and sixth cranial nerves. Orbital pathology such as inflammation, trauma, or neoplasm should be suspected when orbital findings such as chemosis, conjunctival injection, or proptosis are seen.

Isolated third-nerve palsies ("pupil-sparing") are usually caused by microvascular ischemia. Diabetes, hypertension, and advanced age are risk factors. These typically present with intact pupillary function probably because of the superficial location of the pupillomotor fibers.

Management and Disposition

In patients with brainstem involvement, CT or MRI is indicated. Associated fever, headache, and altered consciousness should prompt CT scanning and subsequent spinal tap. MRI with gadolinium is preferred for evaluation of the cavernous sinus, and CT scanning is recommended for suspected orbital pathology.

Of particular concern is the sudden onset of third-nerve palsy accompanied by a "thunderclap" headache, stiff neck, and depressed level of consciousness. Even those with "pupil-sparing" should be evaluated as a neurosurgical emergency with emergent neuroimaging to evaluate for aneurysm and uncal herniation. If subarachnoid hemorrhage is not found and suspicion of aneurysmal leak remains high, a lumbar puncture should be considered.

In the setting of head trauma and oculomotor palsy, the workup should proceed expeditiously, with measures to reduce intracranial pressure.

Pearls

1. Patients with the abrupt onset of a "thunderclap" headache and third-nerve palsy require immediate evaluation for an aneurysm. The posterior communicating artery is a common site.
2. In patients over 50 with third-nerve palsies whose pupil is unaffected ("pupil-sparing"), the etiology is usually hypertensive or diabetic vascular disease.
3. Mild or moderate pain is common in ischemic lesions.
4. Third-nerve palsy is unlikely to cause isolated mydriasis. Other etiologies such as tonic pupil, iris sphincter damage, and pharmacologic mydriasis are more likely.
5. Eighty percent of carotid-cavernous fistulas result from trauma, and may present weeks after minor trauma. Findings include an ocular bruit, pain, pulsatile exophthalmos, and chemosis.

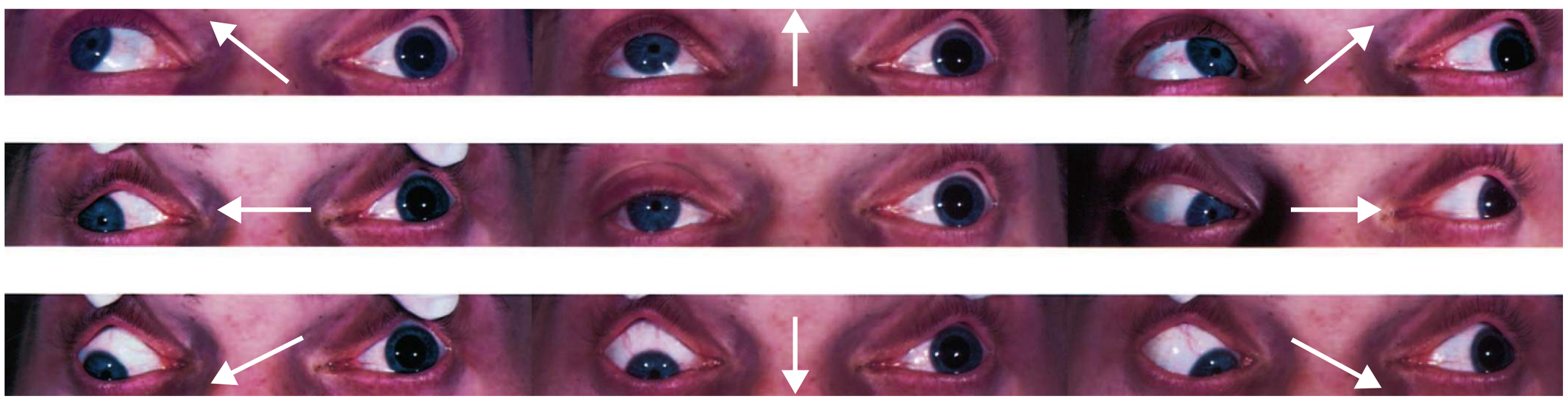

FIGURE 2.59 ■ **Third-Nerve Palsy**. This composite shows the defects of a left third cranial nerve palsy. Conjugate eye movement is present only when the affected eye gazes laterally to the affected side (intact lateral rectus). When gaze is directly ahead, exotropia is seen secondary to the unopposed lateral rectus muscle of the affected side. (Photo contributor: Frank Birinyi, MD.)

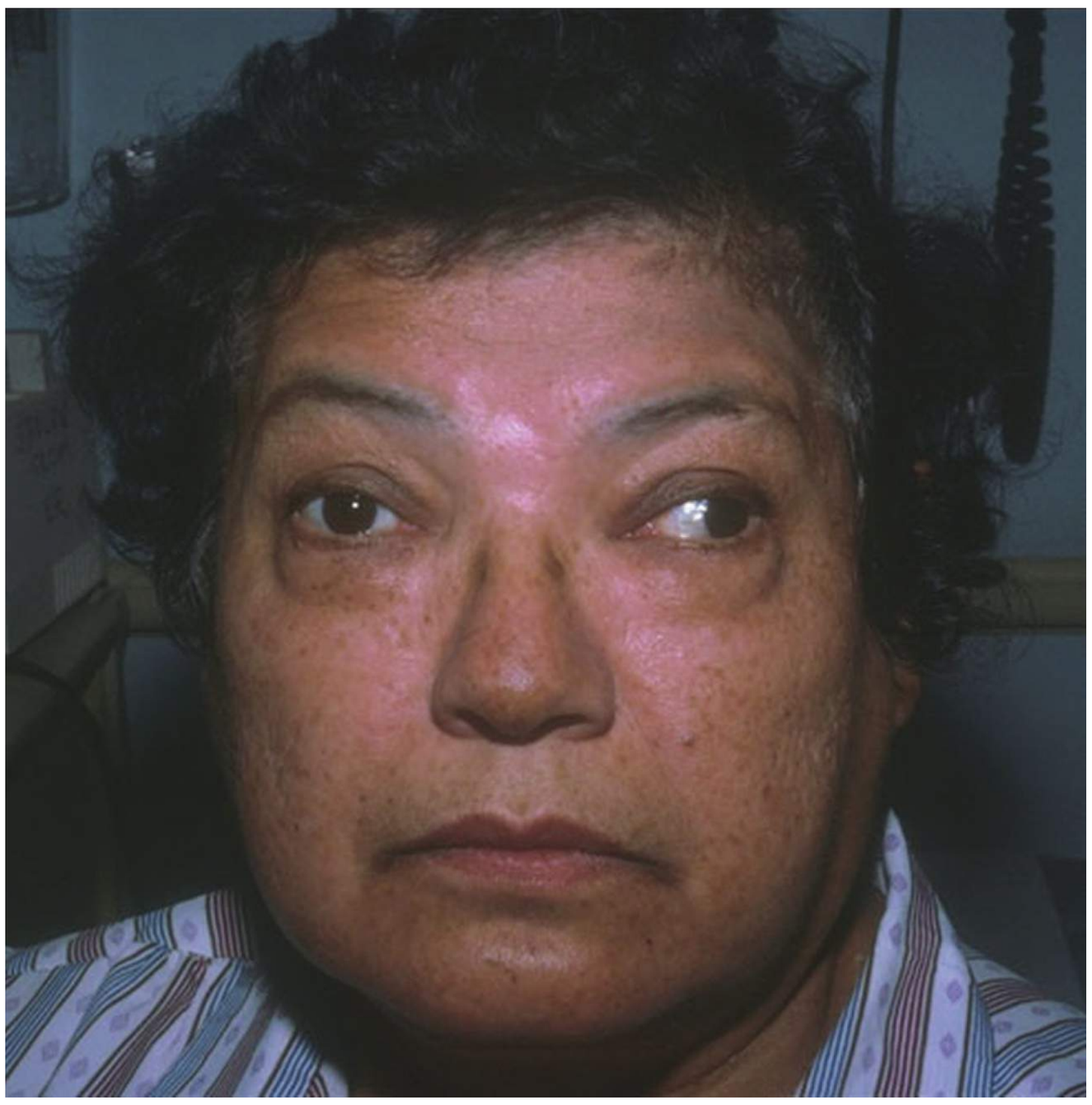

FIGURE 2.60 ■ **Isolated Third-Nerve Palsy**. This 60-year-old woman with diabetes mellitus presents with an isolated third-nerve palsy on the right. She is attempting to look leftward. Her symptoms began as an isolated pain above her right orbit 10 days prior at which time her double vision with leftward gaze began. (Photo contributor: Lawrence B. Stack, MD.)

SIXTH-NERVE PALSY

Clinical Summary

The abducens nerve innervates the lateral rectus muscle and is the most common single muscle palsy, causing loss of abduction and resultant horizontal diplopia, worse in ipsilateral gaze. Associated findings are dependent on the location of the lesion. Within the pons, involvement of the corticospinal tract results in contralateral hemiparesis. The abducens has the longest intracranial course of any nerve, and therefore is vulnerable to stretching or compression secondary to elevated intracranial pressure, trauma, neurosurgical manipulation, and cervical traction. Also, any meningeal process (infectious, inflammatory, or neoplastic) can affect this portion of the sixth nerve. Aneurysmal compression is uncommon.

Prior to entering the cavernous sinus, the nerve crosses the petrous portion of the temporal bone. Trauma with temporal bone fracture can result in a combination of sixth- and seventh-nerve palsies. Cavernous sinus pathology is suggested by the involvement of the internal carotid artery, venous drainage of the eye and orbit, trochlear and oculomotor nerves, the first division of the trigeminal nerve, and the ocular sympathetics. Microvascular changes secondary to diabetes, hypertension, and giant cell arteritis can compromise function.

Management and Disposition

Associated signs and symptoms guide the ED workup. CT or MRI is indicated if brain stem or cavernous sinus involvement is suspected. Pathology localizing to the subarachnoid space should prompt consideration for CT scanning and subsequent spinal tap. In the elderly, an isolated sixth-nerve palsy is likely ischemic, transient, and not indicative of underlying neurologic disease. In these cases, a glucose and erythrocyte sedimentation rate is appropriate; these patients can be followed as outpatients provided close follow-up is arranged.

There is no treatment for the palsy itself except for patching the affected eye if diplopia is bothersome.

Pearls

1. An isolated sixth-nerve palsy is commonly due to microvascular disease, not an aneurysm.
2. Basilar skull fractures of the temporal bone are capable of producing a sixth-nerve palsy.
3. A sixth-nerve palsy associated with a Horner is usually localized to the cavernous sinus, since sympathetic fibers, as they traverse from the internal carotid artery to the oculomotor nerve, may briefly accompany the abducens nerve.

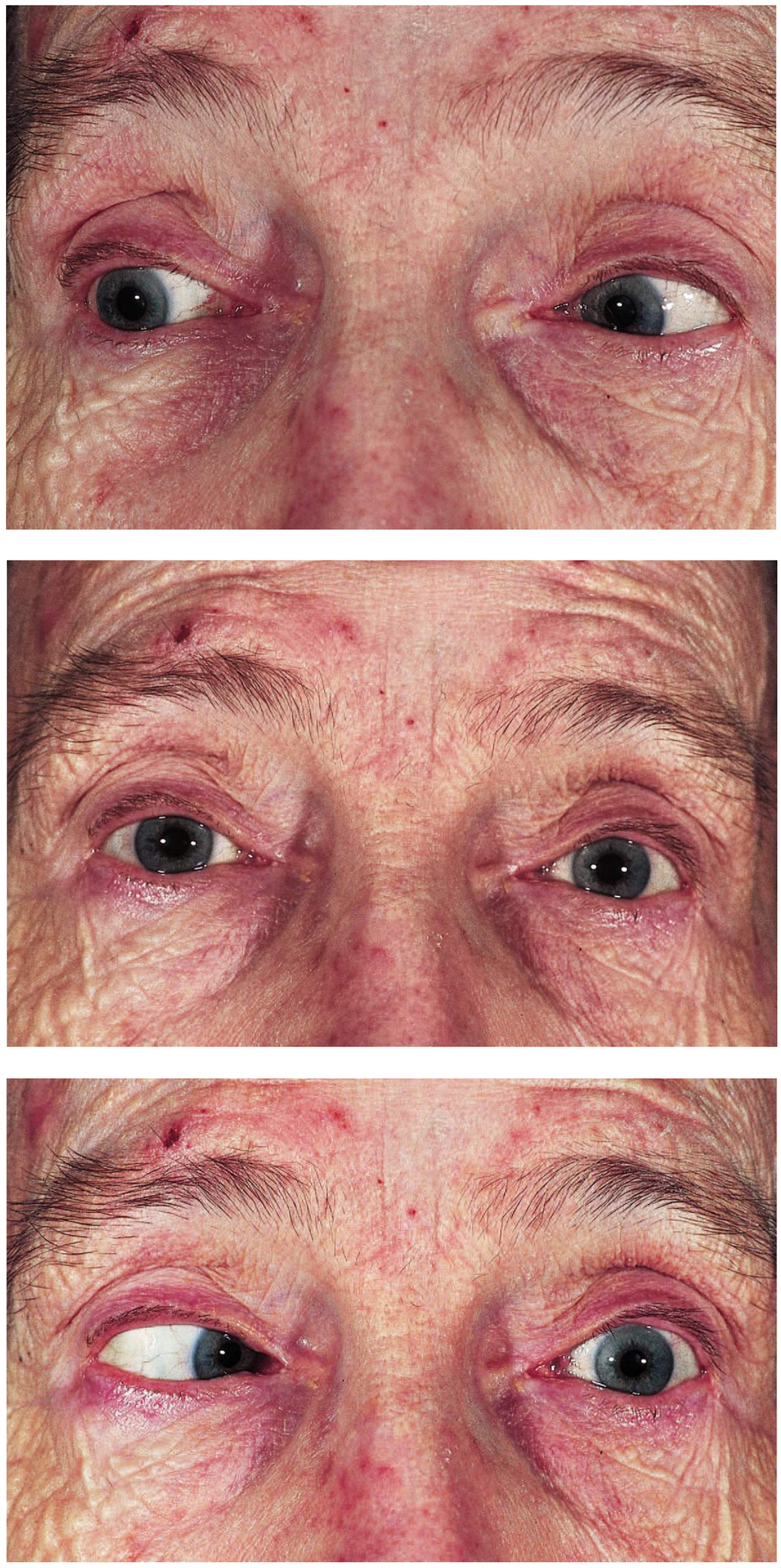

FIGURE 2.61 ■ **Sixth-Nerve Palsy.** Loss of abduction of the left eye is seen in lateral gaze. (Photo contributor: Frank Birinyi, MD.)