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Red eye emergencies in primary care

Abstract: Severe red eye conditions can be the result of intraocular inflammation, corneal insults or inflammation, and acute glaucoma. These pathologies require the knowledge and assessment tools of an ophthalmologist. This article will discuss red eye emergencies that the NP should promptly recognize and refer to ophthalmology.

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Ocular pathologies that require specialized treatment and referral to ophthalmology include: acute angle-closure glaucoma (AACG), globe rupture, uveitis, keratitis, scleritis and episcleritis, and orbital cellulitis.[1,2] A thorough history and physical exam must be performed to differentiate these conditions from common conjunctiva infections and eyelid abnormalities.[3] (See *History and physical exam.*) NPs should maintain a high level of suspicion during the history and physical exam and ask key questions that can lead to the correct diagnosis and management of these ocular pathologies. (See *Physical findings that warrant an ophthalmology referral.*)

AACG

AACG is an uncommon form of glaucoma resulting from the rapid and complete closure of the angle between the iris and the trabecular meshwork of the anterior chamber.[1] This closure is mediated by the anterior displacement of the lens that prevents the outflow of aqueous humor, thereby increasing pressure within the anterior chamber. The rise in intraocular pressure (IOP) is abrupt, causing pain, nausea, and colored halos or rainbows around light.[4] Physical findings are an injected eye with an opaque cornea, IOP greater than 30 mm Hg, shallow anterior chamber, and a characteristic middilated pupil.[5] (See *Acute angle-closure glaucoma.*) Optic nerve damage and blindness can result if untreated, and this condition can occur suddenly as the result moving from an illuminated room to a dark room with dim lighting or in patients taking specific drugs, such as sulfa drugs and topiramate (both drugs cause swelling of the ciliary body).[5]

An immediate referral to ophthalmology is necessary if AACG is suspected. Treatment must be initiated promptly in the primary care office or ED with an oral dose of acetazolamide, and IOP should be measured until the ophthalmologist can assess the patient.[5] Ocular solutions that decrease aqueous humor production and outflow via the trabecular meshwork are then used with one drop of timolol maleate, apraclonidine, and pilocarpine.[5] Each drop is given 1 minute apart and is repeated at 5-minute intervals.[5] Timolol and apraclonidine should be used with caution in patients with chronic obstructive pulmonary disease, asthma, or hypotension.[4] Timely treatment prevents permanent vision loss. Possible complications include repeat episodes, corneal pathologies (edema and cataracts), and iris atrophy.[5]

The American Academy of Ophthalmology (AAO) does not recommend any specific treatment option for AACG.[6] Aqueous humor formation suppression (timolol and acetazolamide) may be ineffective due to ciliary body ischemia from marked IOP.[6] Instead, ophthalmologic surgical intervention (iridotomy or lens removal) may be needed.[6] Per the AAO, the level of evidence to support

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Keywords: acute angle-closure glaucoma, episcleritis, globe rupture, keratitis, orbital cellulitis, scleritis, uveitis

surgical interventions is A:III, which denotes utmost clinical importance with evidence obtained from descriptive studies, case reports, and expert committees and organizations.[6]

Globe rupture

Any eye injury, corneal abrasion, or traumatic orbital wound should alert the NP to the possibility of globe rupture requiring immediate treatment by an ophthalmologist. Globe rupture is the interruption in the integrity of the cornea or sclera, which, if not treated immediately, can cause intraocular infection leading to blindness.[5] Symptoms that alert the NP to globe rupture include: severe pain, decreased visual acuity, hyphema (blood in anterior chamber), loss of anterior chamber depth (aqueous humor escape), and a teardrop-shaped pupil pointing toward the injury (see *Globe rupture*).[5]

Assessing globe rupture requires the Seidel test: fluorescein stain directly to the injury site and wood's lamp exam.[5]

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The Seidel test is positive if the fluorescein stain dilutes within the aqueous, appearing as a darker formation within the bright green fluorescein on wood's lamp exam.[5]

Treatment includes placement of an eye shield to the affected eye and computed tomography (CT) images of the head and orbits (coronal and axial views) to assess for open globe injuries, foreign bodies, or orbital wall fractures.[7] To help prevent increases in IOP, the patient's head should be elevated and the patient should be comfortable. Pharmacologic treatment includes an antiemetic and an analgesic for pain. Antibiotic coverage is based on empiric guidance and should be started with a cephalosporin (such as cefazolin) and a fluoroquinolone (such as ciprofloxacin) I.V. infusion to cover against *Streptococcus*, *Staphylococcus aureus,* and *Staphylococcus epidermidis* species.[5] Alternative regimens include intravitreal (intraocular injection administered by the ophthalmologist) and I.V. antibiotics (such as vancomycin).[8]

Uveitis

Uveitis is an inflammatory process with immune complex deposition within the blood vessels and the structures of the anterior uveal tract.[9] This process is usually associated with systemic autoimmune processes or opportunistic infections within the compromised host. Uveitis has several etiologies: trauma, the human leukocyte antigen (HLA)-B27 genotype (associated with ankylosing spondylitis, Reiter syndrome, inflammatory bowel disease [IBD], and psoriatic arthritis), Behçet disease (triad of uveitis, mouth, and genital lesions), juvenile rheumatoid arthritis, and masquerade syndrome (associated with lymphoma, leukemia, and malignancies of the choroid).[9]

Anterior uveitis has two progressions: granulomatous and nongranulomatous.[9] Nongranulomatous uveitis is not associated with pathologic organisms and is evidenced by small, white keratic precipitates (KPs) with no iris nodules.[9] Granulomatous uveitis follows a microbial infection (cytomegalovirus, tuberculosis, syphilis, and toxoplasmosis) and is associated with large, mutton-fat KPs and iris nodules.[9]

History and physical exam[1]

History

- Onset: sudden or gradual?
- Other family members affected with same symptoms?
- Is the patient using any medications?
- Was there any trauma to the eye?
- Is one (both) eye(s) affected?
- Does the patient use contact lenses, and did the patient sleep in the lenses?
- Did the patient have recent eye surgery?
- Is there decreased vision or pain?
- Is there any discharge from the affected eye(s)?
- Is the discharge scant, profuse, watery, or purulent?
- Does the eye itch?
- Is there sensitivity to light?
- Are there any other symptoms associated with the eye?

Physical exam

- Visual acuity
- Confrontation testing (peripheral vision)
- Adnexal assessment of lids, lashes, and surrounding tissues
- Assessment of the sclera and conjunctiva
- Extraocular movements
- Testing of pupils for direct and consensual responses
- Inspection of the cornea and iris
- Assessment of the anterior chamber (penlight and slit-lamp evaluation)
- Fluorescein stain if corneal integrity is compromised
- Lens assessment for clarity
- Fundoscopic exam
- Tonometry

Physical findings that warrant an ophthalmology referral[1]

Reduced visual acuity
Occurs with serious ocular disease, such as an inflamed cornea, iritis, or glaucoma. Reduced visual acuity never occurs in simple conjunctivitis.

Ciliary flush
A pattern of injection in which the redness is most pronounced in a ring at the transition zone between the cornea and the sclera (limbus), found in uveitis, corneal inflammation, or acute glaucoma.

Photophobia
Unusual sensitivity to light that can signify uveitis or corneal inflammation/injury.

Severe pain/foreign body sensation
Inability to keep the eye open, indicating corneal inflammation or ulceration, uveitis, scleritis, or glaucoma.

Corneal opacity
Keratic precipitates or cellular deposits on the cornea within anterior chamber suggestive of uveitis. Diffuse haze covering cornea suggestive of edema (acute glaucoma).

Corneal injury
Found on fluorescein stain (bright green area) or an irregular light reflex on penlight exam.

Fixed pupil
Uveitis typically presents with one pupil smaller than the other due to reflex spasm of the iris muscle and/or inflammatory adhesions (keratic precipitates) to the site. Acute glaucoma presents with single, fixed/middilated pupil that is slightly irregular.

Headache and nausea
Indicates acute angle-closure glaucoma.

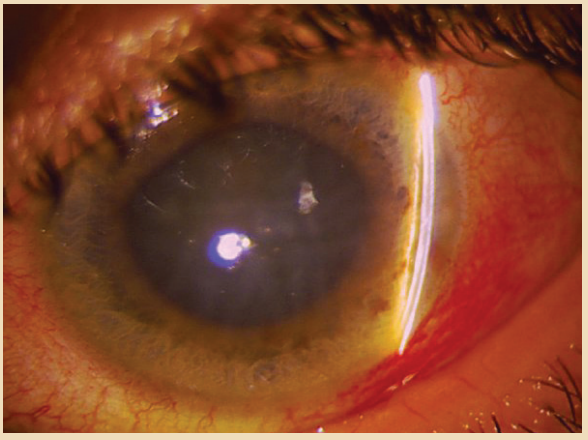
Purulent discharge and hyperemia
Suggestive of gonococcal conjunctivitis.

Shallow anterior chamber
Shine penlight from temporal side of eye in a plane parallel to iris, looking at nasal side of iris, if two-thirds or more of nasal iris is in shadow, the chamber is probably narrow.

Increased IOP
Mean IOP is 15 mm Hg (range: 10-21 mm Hg), elevations above these measurements signify increased IOP: acute angle glaucoma or iridocyclitis.

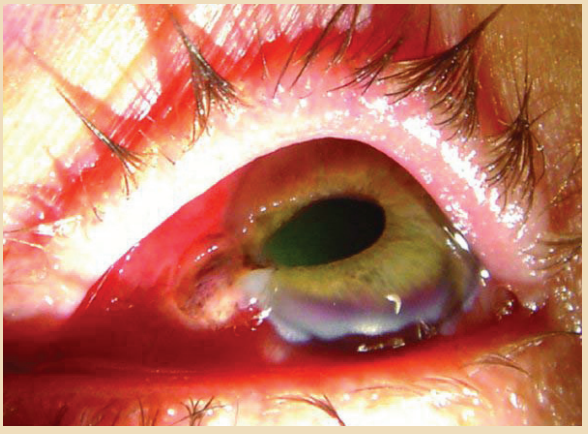
Proptosis with painful EOM
Forward displacement of the globe, suggestive of periorbital and cavernous sinus disease. Best assessed by tilting chin up and assessing orbits inferiorly from the chin.

Acute angle-closure glaucoma



Source: Gerstenblith AT and Rabinowitz MP. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease.* 6th. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.

Globe rupture



Source: Gerstenblith AT and Rabinowitz MP. *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease.* 6th. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.

Patients with anterior uveitis present with: ciliary flush, unilateral pain, redness, photophobia, and acute vision loss.[1] Nongranulomatous uveitis symptoms progress acutely, with circumferential erythema (ciliary flush) from the increased permeability of the uveal vessels.[9] Uveitis results in inflammatory cells within the anterior chamber that produce the "cell and flare" within the aqueous when examined via the slit lamp.[9] KPs can be seen via penlight and magnifying lens implanted on the corneal epithelium.[9] (See *Uveitis.*)

Treatment must be initiated by the ophthalmologist and usually takes the form of topical and systemic corticosteroids.[1]

Anterior segment inflammation is typically treated with dexamethasone sodium phosphate 0.1% solution.[10] Cholinergic agonists such as atropine 0.5% to 1% eye drops are used to block the neurotransmission sites in the iris sphincter, which relieves pain by immobilizing the iris, preventing adhesions of the iris, and stabilizing the blood-aqueous barrier to prevent further leakage of cellular material.[9]

Keratitis

Keratitis is inflammation and/or infection of the cornea, which stems from infectious (bacterial, viral, fungal, or protozoal) or noninfectious (chemical injuries, dry eyes, inflammatory disorders, or severe allergies) causes.[11] Patients with corneal ulcerations present with hyperemia, mucous secretions within the anterior chamber (hypopyon), and corneal opacities.[1] Patients report pain, photophobia, tearing, and decreased vision.[12]

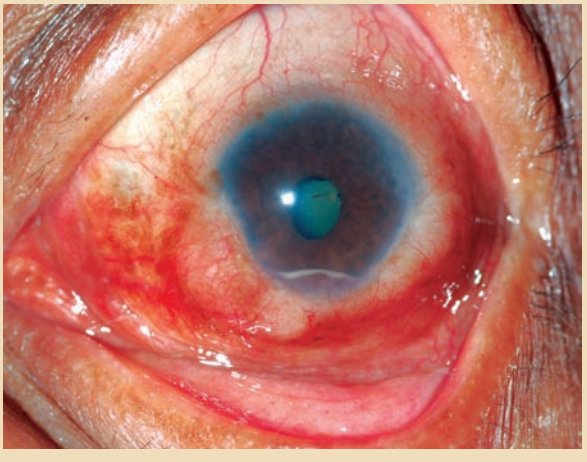
Bacterial infections cause epithelial ulceration from cytokine infiltration reaching Bowman's layer (basement membrane).[13] Corneal opacity (typically a round, white spot) is the clinical evidence of bacterial keratitis in association with red eye, photophobia, and foreign body sensation.[13] These findings are confirmed with fluorescein stain and penlight exam. (See *Bacterial keratitis*.) Common causes include trauma, recent surgery, and contact lens use (especially overnight use).[13] The most common organisms involved include *S. aureus* (MRSA), *Pseudomonas aeruginosa, Pneumococcus, Moraxella,* and staphylococci species.[13] Treatment includes the administration of fluoroquinolone eye drops.[14]

Fluoroquinolone eye drops are given an evidence rating of A:I, noting that they are of key clinical importance and derived from meta-analysis of randomized controlled trials with a low-risk bias.[15] Alternatives to fluoroquinolone therapy include tobramycin solution.[15] Referral to an ophthalmologist is indicated to ensure proper management.

Gonococcal conjunctivitis is a form of conjunctivitis that can be sight threatening.[13] Transmitted through hand-genital-eye contact, the bacteria spreads rapidly and can easily penetrate the corneal surface.[13] Symptoms include: profuse purulent ocular discharge (greater than other forms of conjunctivitis), conjunctival chemosis, irritation, preauricular lymphadenopathy, and tenderness to palpation within 12 hours of infection.[13] There may or may not be concurrent urethritis. If left untreated, corneal perforation and melting can occur, which makes this a medical emergency.[13] Treatment includes an initial I.M. injection of ceftriaxone and saline lavage.[13] Alternative treatments include azithromycin as a single-oral dose or doxycycline therapy for 7 days.[15] These treatments are endorsed by AAO (A:II), which evidences clinically-significant, high-quality systematic reviews of case-control cohort studies.[15]

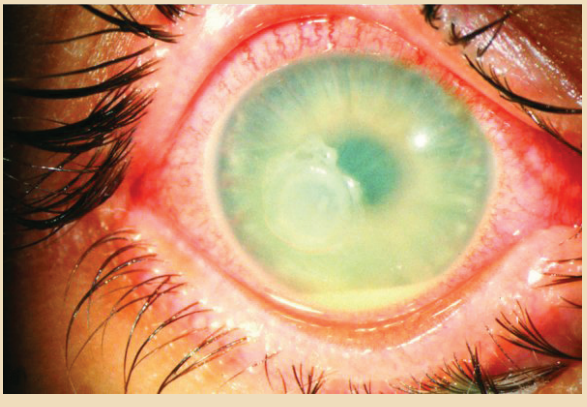
Herpes keratitis takes the form of herpes simplex (HSV) and herpes zoster (HZV) ocular infections. Viruses colonize the trigeminal nerve ganglion, which lead to concurrent reinfections.[14] Outbreaks occur with fevers, exposure to sunlight, stress, or immunocompromised states.[14] Patients typically present with fever, malaise, headache, and periocular burning, which herald a vesicular, then pustular, then crusting confluent ulceration to the trigeminal dermatome.[16] Patients may report history of HSV infection, and exam reveals watery discharge, ciliary flush, and decreased visual acuity.[16] Fluorescein staining reveals corneal punctate keratitis with both HSV and HZV; however, HSV merge to form the characteristic branching "dendritic" ulceration.[16] Ocular

Uveitis



Source: Garg SJ. *Color Atlas and Synopsis of Clinical Ophthalmology Wills Eye Institute – Uveitis*. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.

Bacterial keratitis



Source: Rapuano CJ. *Color Atlas and Synopsis of Clinical Ophthalmology Wills Eye Institute – Cornea*. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.

signs of viral infection include conjunctivitis, episcleritis, and uveitis.[14]

Treatment for corneal HSV infections includes corneal debridement, antiviral therapy with trifluridine eye drops until reepithelialization occurs (21 days).[14] Ganciclovir 0.15% ophthalmic gel 1 drop 5 times daily until ulceration heals, then 3 times daily for 7 days.[17] Corticosteroid eye drops, prescribed by the ophthalmologist, are usually added to the treatment regimen.[11] Treatment of HZV infection also includes high-dose oral acyclovir therapy.[14] Alternative treatments to oral therapy are indicated if the patient has had multiple HSV keratitis infections indicating possible acyclovir resistance.[17] Systemic treatment with foscarnet is advised if resistance is suspected.[17] AAO supports these treatment regimens with level A:I evidence, indicating systematic reviews of randomized controlled trials.[17]

Chemical injuries are another source of emergent corneal injury, with alkali substances being more injurious than acidic contacts.[5] The injury's severity is variable depending on the pH concentration and chemical nature of the solution.[5] Photophobia, foreign body sensation, severe pain, and blurred vision ensue almost instantaneously after exposure to the chemical.[5] Patients will constantly blink (reflex blepharospasm) in almost all chemical injuries.[5] Severe alkali exposures cause conjunctival and scleral ischemia, causing a "porcelainized" corneal appearance.[5] (See *Chemical burn*.)

A visual acuity exam should not be performed in this setting, and immediate irrigation should ensue. Ocular pH should be tested by placing pH paper between the eyelid and the globe; normal ocular pH is "neutral" between 7.0 and 7.3.[5] This pH measurement is used to guide therapy. Before irrigation, an ocular anesthetic such as tetracaine should be instilled and a Morgan lens placed to irrigate with 0.9% sodium chloride or lactated Ringer's solution.[5] Irrigation should continue until pH returns to 7.0.[5] An antibiotic such as ofloxacin drops can be instilled after stabilization occurs to prevent infection.[5]

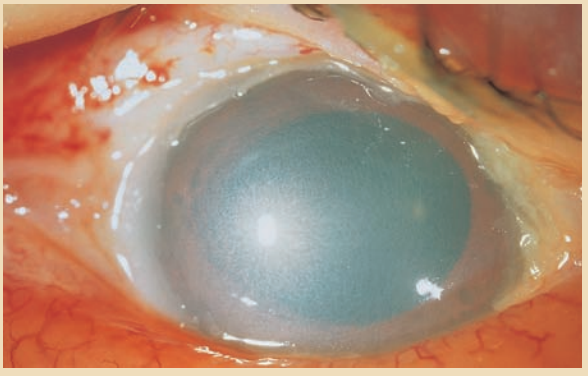
Scleritis and episcleritis

Scleral inflammation comes in the form of episcleritis (superficial) and scleritis (deeper and more destructive) inflammation.[18] Both processes are usually associated with systemic autoimmune processes; however, episcleritis is more benign and can be the result of dry eye and viral infections.[18] Approximately 39% to 50% of cases are associated with systemic disease, with the most common tissue disorders being rheumatoid arthritis and Wegener granulomatosis.[19] Scleritis carries more ocular risks, including keratitis, uveitis, and glaucoma.[18] The majority of patients have underlying autoimmune diseases, such as rheumatoid arthritis, IBD, or systemic lupus erythematosus.[18]

Immunoglobulin E degranulation, systemic vasculitic diseases (tuberculosis and syphilis), and granulomatous diseases are the causes of immune-mediated episcleritis.[18] Episcleritis presents with bilateral ocular pain and peripheral injection pattern with minimal lacrimation or photophobia.[18] (See *Episcleritis*.)

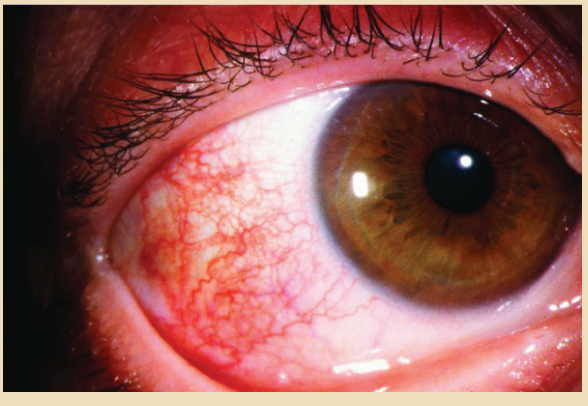
Scleritis presents with a deeper red injection to all layers of the sclera and globe, edema, and ocular tenderness.[20] (See *Scleritis*.) Scleritis can lead to necrotizing scleral ulceration and perforation; therefore, ophthalmologic consult is required.[20] Instillation of topical phenylephrine is utilized to differentiate scleritis from episcleritis or conjunctivitis. If the diagnosis is episcleritis or conjunctivitis, hyperemia clears;

Chemical burn



Source: Chern KC, Saidel MA. *Ophthalmology Review Manual.* 2nd. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.

Episcleritis



Source: Rapuano CJ. *Color Atlas and Synopsis of Clinical Ophthalmology Wills Eye Institute – Cornea.* 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.

however, if deeper layers are affected, scleral erythema will not clear, and scleritis should be suspected.[18]

Treatment of episcleritis involves polyethylene glycol 400 and propylene glycol eye drops as needed, oral nonsteroidal anti-inflammatory drugs (NSAIDs), and topical corticosteroids as needed.[18] Scleritis involves the use of NSAIDs and oral prednisone initially.[18] Bolus or pulse therapy with I.V. glucocorticoids may be given followed by oral prednisone.[21] Ophthalmologic referral is necessary for both to ensure proper diagnosis and management.[18]

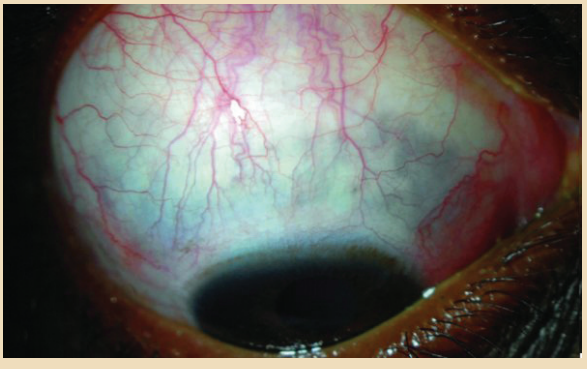
Orbital cellulitis

Orbital cellulitis is an infection that involves the orbit (fat and ocular muscles).[22] It is differentiated by preseptal (infection involving tissue anterior to the orbital septum) and periorbital (infection of tissue posterior to the septum).[22] (See *Preseptal cellulitis*.) Orbital cellulitis stems from unresolved ethmoid sinus infection, orbital trauma, and ocular surgery.[22] Preseptal cellulitis stems from: sinusitis, soft tissue infection of face or eyelid, trauma, insect bites, and foreign bodies.[22] Bacterial causes include staphylococcus species *S. aureus, S. epidermidis,* and the following streptococcus species: *Streptococcus anginosus, Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis,* and group AB-hemolytic streptococci.[23] Without proper and rapid treatment, preseptal presentations can result in orbital infections, and orbital infections can cause blindness, meningeal infections, optic nerve involvement, and cavernous sinus thrombosis.[22] Patients with preseptal cellulitis present with lid swelling, conjunctival congestion, and pain. Patients with orbital cellulitis present with the same symptoms; however, they also have proptosis, increased IOP, and pain with extraocular movements (EOM).[22]

Empiric treatment of preseptal cellulitis includes oral clindamycin alone or in conjunction with amoxicillin-clavulanate or cefpodoxime twice daily with close follow up.[22] Orbital cellulitis requires CT imaging, hospitalization, and I.V. antibiotics, including: vancomycin, ceftriaxone, and metronidazole.[22] Alternative treatment regimens include inpatient versus outpatient antibiotic therapy.[24] A referral to the ophthalmologist is indicated to ensure that there are no intraocular pathologies associated with the orbital infections.[22]

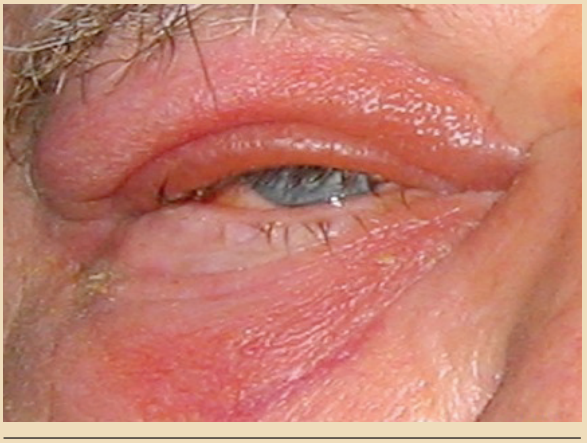
An increased suspicion and rapid recognition of these red eye emergencies by nurse practitioners in all practice settings will help ensure improved patient outcomes with the best chances to preserve eyesight. Although many ocular conditions can be treated in the primary care office, there are these select cases that require specialist consultation. Primary care NPs should never hesitate to call on the experience and knowledge of a trusted ophthalmologist to ensure proper patient care. NP

Scleritis



Source: Rapuano CJ. *Color Atlas and Synopsis of Clinical Ophthalmology Wills Eye Institute – Cornea.* 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012.

Preseptal cellulitis



Source: Dinn RB, Graff M. *Preseptal cellulitis.* University of Iowa Health Care: Opthalmology and Visual Sciences. Updated February 2, 2010. Used with permission from EyeRounds.org and The University of Iowa: www.EyeRounds.org.

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