Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Volume 32, Number 15 / July 4, 2011 www.emreports.com

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AHC Media

The Red Eye

Introduction

The number of adult ED visits for eye-related complaints is largely limited to data on eye injuries.[1,2] According to the latest National Health Statistics Report, 1.2 % of all injury-related visits to emergency departments (EDs) in the United States in 2007 were for eye injuries.[1] A recent analysis showed that infectious and non-infectious diseases of the eye accounted for 1.6% of ED pediatric visits in 2002.[3] Given the diversity of non-traumatic ocular pathology, this likely represents a gross underestimation of the number of patients presenting with complaints related to the eye.

Most patients presenting to the ED with eye complaints have a red eye. The differential diagnosis includes a broad range of local infectious, inflammatory, and traumatic processes, as well as systemic diseases that affect the globe. Patients must be evaluated expeditiously if a potential ocular emergency exists, such as a chemical injury. Clues to potentially serious ocular pathology are eye pain, visual disturbance, eye trauma, or a patient in distress; these patients must be given a high triage priority. This article will discuss the more important inflammatory causes of red eye. Determining the exact etiology or causative agent may not be possible. In fact, many of the conditions discussed have wide overlap in symptoms, etiologic agents, and associated systemic diseases. In addition, certain disease states can present with a spectrum of ocular conditions. For example, systemic lupus erythematosus may present with conjunctivitis, scleritis, keratitis, or uveitis. Eye trauma, although an important consideration in the evaluation of red eye, will not be discussed in this article.

Ocular Anatomy and Pathophysiology

The eyeball is divided into three layers or "tunics": the outer fibrous tunic, the middle vascular tunic, and the inner neural tunic. The outer layer of the eyeball, the fibrous tunic, is a collagenous membrane that is composed of the sclera and the cornea.

The middle layer of the eye is the vascular tunic, which is composed of the choroid posteriorly and the iris and ciliary body anteriorly. The choroid is rich in vasculature and pigment. The ciliary body is composed of the ciliary processes that secrete aqueous humor and the ciliary muscle that attaches to the lens.

The retinal or nervous tunic forms the innermost layer of the eyeball and contains the photoreceptors.

Aqueous and Vitreous Humor. The vitreous humor is located in the posterior chamber and the aqueous humor is located in the anterior chamber. Small changes in the production or drainage of aqueous humor have a big effect on intraocular pressure. A significant increase in intraocular pressure can compress the optic nerve in the posterior portion of the eye and cause visual loss.

Initial Approach to the Patient with Red Eye

History. Important data include past ocular diseases or procedures, a

Executive Summary

- Chemical injuries to the eye with acids or alkali should be treated with immediate irrigation.
- Episcleritis is a benign disease that resolves spontaneously. Scleritis is a more serious disease.
- A slit lamp exam reveals flare and cells in the anterior chamber in anterior uveitis.
- Acute angle glaucoma has elevated pressures in the eye as well as decreased vision. Patients may have abdominal pain.

complete medical history, and a history of prescription and nonprescription medication use. The ED physician should determine the duration of symptoms and the presence or absence of certain indicators of eye pathology such as pain, discharge, change in visual acuity, contact lens use, photosensitivity, trauma, or foreign body exposure.

Examination of the Eye. Visual acuity testing is done bilaterally and is generally done first. The patient should wear his or her corrective lenses if available. Pinhole visual acuity testing is performed if the patient's corrective lenses are unavailable or when visual acuity is abnormal to correct for possible refractive errors. Check the pupils for irregularity in size or reactivity and photophobia. The eye should then be examined from the outside in, starting with the lids and lashes. Evert the lid to look for a conjunctival foreign body. Perilimbic conjunctival injection ("ciliary flush") is indicative of more serious pathology such as acute iritis or acute glaucoma. The cornea should be examined for abrasions and ulcerations using fluorescein eye stain and a blue light source. A slit-lamp examination should be performed to magnify the external ocular structures and anterior chamber. The anterior chamber should be assessed for depth and the presence of inflammatory cells or blood. Unless a ruptured globe is present or suspected, ocular pressure should be measured on the anesthetized eye.

Simple causes of red eye are easily treated in the ED. Consult an ophthalmologist when serious complaints exist or if the diagnosis is uncertain and there are symptoms of more serious ocular pathology, as listed above or for any condition that warrants topical steroids.

Inflammatory and Infectious Causes of Red Eye

Conjunctivitis. Conjunctivitis, or inflammation of the conjunctiva, is the most common cause of red eye. Vascular dilatation, cellular infiltration, and exudation are the hallmark of the disease. The conjunctiva is divided into the bulbar conjunctiva, which covers the globe, and the tarsal conjunctiva, which lines the eyelids. Conjunctivitis may involve one or both of these structures.

The etiology of conjunctivitis is usually attributed to bacteria and viruses, but may also be due to allergens and other irritants, and less commonly to fungus, parasites, and systemic disease. For simplicity, conjunctivitis is often classified as infectious or non-infectious. Infectious causes are further subdivided into bacterial or viral, and non-infectious causes are subdivided into allergic and non-allergic.

The presenting complaint in conjunctivitis is acute, diffuse conjunctival injection that rapidly spreads from one eye to the other, and eye discharge. Eyelid edema may or may not be present. The diagnosis of simple conjunctivitis should be questioned if the patient presents with eye pain, photophobia, decreased visual acuity, an abnormally reactive pupil, increased intraocular pressure, corneal opacification, or localized conjunctival injection. These complaints should alert the physician to look for other more serious eye pathology. Overall, conjunctivitis is a benign, self-limited disease that rarely causes severe sequelae. It is a clinical diagnosis in most settings, and cultures are rarely taken. Distinguishing between the different types of conjunctivitis based on clinical presentation is not reliable.[4]

Infectious Conjunctivitis.

Bacterial Conjunctivitis. Nearly 4 million cases of bacterial conjunctivitis were seen in the United States in 2005, with direct and indirect costs estimated at nearly $600 million.[5] Because conjunctival cultures are rarely obtained, the true incidence of this disease may be misrepresented by available data. Disruption in the epithelial surface allows pathogens to infect the conjunctiva and cause an inflammatory reaction. Infection in surrounding structures, such as obstructed nasolacrimal ducts, pharyngitis, bacterial sinusitis, or otitis media, may spread to the conjunctiva. In addition, disorders of the lids or tear film production may inhibit the normal protective barrier and make the patient susceptible to infection. The clinical presentation usually begins with unilateral conjunctival injection, mucopurulent discharge, and eyelid edema that rapidly progresses to bilateral eye involvement in 1 to 2 days. Patients may experience a "gritty" sensation in their eyes, but true pain is unusual. Common pathogens are *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Haemophilus influenzae*, *Streptococcus pneumoniae*, and Moraxella species.[6] Different bacterial pathogens predominate in different age groups and with different acuity.

Neonatal conjunctivitis (ophthalmia neonatorum) occurs within the first 28 days of life. Many cases are

acquired by passage through the birth canal. In the United States, it occurs in 1-2% of births and is most often caused by *Chlamydia trachomatis*, *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Escherichia coli*, *Neisseria gonorrhea*, other gram-negative bacteria, and herpes simplex virus.[7] *Chlamydia trachomatis* is the most common cause, but *Neisseria gonorrhea* is the most destructive and must be excluded in all cases. Neisseria conjunctivitis typically presents earlier, in the first 24-48 hours of life, while Chlamydia conjunctivitis manifests later, usually at 1-2 weeks. Neonates also get conjunctivitis as the result of a blocked tear duct. Because topical silver nitrate instillation at birth is no longer a common procedure, chemical conjunctivitis is less common.

Childhood conjunctivitis is predominantly caused by *Streptococcus pneumoniae* and *Haemophilus influenzae*. Large outbreaks may occur in daycare or school settings.

Adult conjunctivitis is usually caused by Staphylococcus species.

Acute conjunctivitis is the most common type of bacterial conjunctivitis and usually lasts less than 4 weeks. Chronic bacterial conjunctivitis is present if the infection lasts longer than 4 weeks. In this circumstance, symptoms are often mild but recurrent, and infection is usually caused by coagulase-positive and -negative staphylococci.[8]

Hyperacute bacterial conjunctivitis deserves special mention because it is an aggressive form of conjunctivitis that may progress rapidly to involve the cornea and intraocular cavities. It is primarily caused by *Neisseria gonorrhea* and is most often seen in neonates and young sexually active adults. In adults, a concurrent genital infection is usually present, but may be asymptomatic. Symptoms occur within 24 hours and present as a painful hyperemic conjunctiva, profuse mucopurulent discharge, marked conjunctival chemosis, and preauricular adenopathy.

Routine culture is not indicated for simple bacterial conjunctivitis. Cultures should be performed in neonates, immunocompromised patients, and in those with severe acute conjunctivitis or hyperacute conjunctivitis when gonorrhea is suspected. It should also be considered in chronic or recurrent conjunctivitis, or those cases not responding to treatment. Bacterial conjunctivitis has an extremely high rate of spontaneous remission and rarely causes serious ocular sequelae. Because bacterial conjunctivitis is highly contagious, local hygienic practices, such as frequent hand-washing, must be encouraged. It is established medical practice to treat acute bacterial conjunctivitis with broad-spectrum topical antibiotics, although there is significant literature to suggest that no treatment or delayed treatment yields similar outcomes.[9-14] The choice of which agent to use does not usually affect outcome.[15] Topical fluoroquinolones, trimethoprim/polymyxin B, sulfacetamide, erythromycin, and gentamicins are among the available preparations used. Topical azithromycin ophthalmic solution was approved in 2007 for use in bacterial conjunctivitis, with the advantage of once- to twice-daily dosing. Reports show similar efficacy to other currently available preparations.[16,17]

Adult gonococcal conjunctivitis is treated with single-dose intramuscular ceftriaxone, plus or minus topical antibacterials. In neonatal conjunctivitis, gonococcal infection is treated with single-dose ceftriaxone, 100 mg/kg IV or IM. Pediatric chlamydial infection is treated with erythromycin 50 mg/kg PO 4 times daily for 14 days.[18] Topical medication alone is not effective in either gonococcal or chlamydia conjunctivitis of the newborn, and not necessary when systemic treatment is administered.

Viral Conjunctivitis. Viral conjunctivitis is classically described as having a watery discharge, an acute follicular conjunctival reaction, preauricular lymphadenopathy, and a longer clinical course than bacterial conjunctivitis, often lasting several weeks. Viral conjunctivitis presents as an isolated conjunctivitis or as part of a generalized viral syndrome, especially in children. While a number of viruses cause viral conjunctivitis, adenovirus is the most common. Other causes include enterovirus, varicella zoster virus, herpes simplex virus (HSV), Epstein-Barr virus, poxviruses, and coxsackievirus. Adenovirus and HSV are the two viruses discussed in detail here; the former because of its prevalence, and the latter because of its potential for serious complications if misdiagnosed.

Adenovirus has many different serotypes, 19 of which cause conjunctivitis.[19] It is extremely contagious, usually through upper respiratory droplets or finger to eye contact during the first few days of symptoms. Adenovirus presents as one of several well-described syndromes: as a follicular conjunctivitis, as pharyngoconjunctival fever, or as epidemic keratoconjunctivitis. They are often indistinguishable early in the disease course. The mildest form is follicular conjunctivitis, which usually presents with mild, unilateral conjunctival injection, watery discharge, follicular and papillary conjunctival reactions, and preauricular lymphadenopathy on the involved side. As the name implies, pharyngoconjunctival fever presents as a febrile illness with pharyngitis and conjunctivitis, and is often confused with influenza. It is most commonly seen in school-age children. A follicular conjunctival reaction is seen with watery discharge, conjunctival injection, chemosis, and preauricular lymphadenopathy in the majority of cases.

Epidemic keratoconjunctivitis (EKC) is a more severe, contagious form of conjunctivitis that can involve the cornea as well. It is associated with sporadic outbreaks and persistent infection. EKC is usually a bilateral follicular conjunctivitis accompanied by pain and photophobia, characteristics less commonly seen in other viral conjunctivitis. Preauricular adenopathy and serous discharge are common, but the hallmark of the disease is the presence of conjunctival membranes (often termed "pseudomembranes"),

epithelial and subepithelial corneal infiltrates, and preauricular lymphadenopathy. The membranes are most common on the tarsal plates and may be missed on examination. The corneal infiltrates seen in EKC are present in only about one-third of the patients.[19] Central corneal infiltrates, or ones that coalesce, may reduce visual acuity.

HSV conjunctivitis (HSVC) occurs more often in children and adults and is most often caused by herpes simplex virus type 1 (HSV-1). When it occurs in neonates, it is usually caused by herpes simplex virus type 2 contracted during passage through the birth canal. It causes a papillary conjunctivitis that is often accompanied by eyelid infection, and is similar to adenovirus conjunctivitis.[20] Signs that distinguish this infection from adenovirus are its tendency to be unilateral, the presence of a vesicular or ulcerating rash on the eyelids, and dendritic epithelial disruption of the cornea. It is the most common cause of blindness in third-world countries. There are about 500,000 cases diagnosed in the United States each year.[21]

Herpes zoster ophthalmicus (HZO, or "ocular shingles") is caused by the varicella zoster virus (VZV). Immunocompromised patients are at higher risk for developing this disease. Vesicles on the tip of the nose are a clue to this disorder and are the result of nasocilary (V1) nerve involvement (Hutchinson's sign). Since this nerve branch also innervates the cornea, patients with Hutchinson's sign and conjunctival injection must be suspected of having ocular involvement. A small number of patients may have eye involvement without the characteristic rash, and ocular involvement can occur with a rash anywhere on the forehead. While conjunctivitis itself is usually self-limiting, secondary bacterial superinfection may occur. Any patient who presents with herpetic vesicles in the distribution of the trigeminal nerve should undergo examination of the conjunctiva, cornea, and anterior chamber. In a recent study in Academic Emergency Medicine, eye redness in patients with HZO was found to be 100% predictive of moderate to severe eye disease. They recommend ophthalmology consultation in these patients, but not necessarily in patients without eye redness, even in the presence of Hutchinson's sign.[22]

Potential complications of adenovirus and herpes conjunctivitis include conjunctival scarring and involvement of the cornea or deeper ocular structures, with the possibility of vision loss. For adenovirus conjunctivitis, there is no effective antiviral medication and treatment is aimed at relief of symptoms. Cool compresses, vasoconstrictive eye drops, and artificial tears may ease symptoms. If bacterial superinfection exists, a broad-spectrum topical antibiotic should be prescribed. Careful hygiene is warranted to prevent the spread of infection to others. In severe cases, topical steroids may be prescribed to decrease symptoms and minimize scarring, but this should be done by the consulting ophthalmologist. In EKC with pseudomembrane formation, debridement of the membrane may provide increased comfort to the patient.

HSV conjunctivitis may be treated with antiviral medication to prevent infection from spreading to the cornea. Oral (acyclovir, valacyclovir, famciclovir) or topical (ganciclovir gel, trifluridine solution, vidarabine ointment) antivirals can be used. Topical antivirals may produce epithelial toxicity. They should be tapered rapidly as the patient improves, and be limited to a two-week course of treatment. Topical antibiotics are given to prevent bacterial infection. Neonatal ocular herpes infection requires prompt infectious disease consultation due to the high incidence of systemic and neurologic disease in this age group. In VZV infections, the oral antivirals listed previously are helpful to prevent ocular involvement if given to patients with cutaneous involvement only in the first three days from onset of the rash. Topical antibiotics may be given to prevent infection. Topical antivirals have not been shown to be effective once conjunctivitis is diagnosed. Topical steroids are indicated in the treatment of herpetic stromal keratitis, which involves the middle, transparent layer of the cornea most susceptible to scarring and subsequent visual loss. When this diagnosis is suspected, ophthalmology consultation is essential.

Non-infectious Conjunctivitis. *Allergic Conjunctivitis.* Four types of allergic reaction are described: seasonal and perennial allergic conjunctivitis (SAC and PAC), vernal keratoconjunctivitis (VKC), atopic keratoconjunctivitis (AKC), and giant papillary conjunctivitis (GPC). Each of these may present a similar initial picture, with bilateral findings of eyelid edema, conjunctival injection and edema, watery discharge, and itching or burning in the affected eyes. Purulent discharge is uncommon. The patient with allergic conjunctivitis will often have a personal or family history of atopy. The presence of nasal congestion, sneezing, and rhinorrhea with conjunctivitis (rhinoconjunctivitis), by history or on physical examination, is a clue to the allergic nature of the patient's symptoms.

Seasonal and Perennial Conjunctivitis. Ocular allergies are usually due to SAC and PAC. They are acute, mild, and uncomplicated. SAC predominates during the spring to fall months when airborne allergens are abundant. Patients with PAC have symptoms year-round. Environmental, animal, and household allergens are thought to trigger their symptoms.

Vernal and Atopic Keratoconjunctivitis. These two types of ocular allergy represent approximately 2% of allergic conjunctivitis.[23] Chronic inflammation is believed to involve different cytokines and effector cells such as eosinophils and basophils.[24,25] Patients frequently have a personal or family history of atopy, such as asthma, eczematous dermatitis, or allergic rhinitis. VKC and AKC are more chronic, recurrent, and potentially serious processes. The complications seen with both of these diseases are due to corneal

Figure 1: Large Papillae with a Classic "Cobblestone" Appearance Seen in Vernal Keratoconjunctivitis

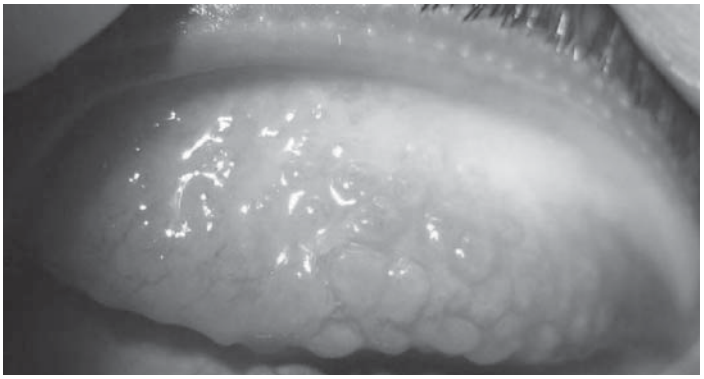


Image used with permission from: Dr. Sunil Kumar.

involvement and the potential for visual loss secondary to corneal scarring. VKG predominates in childhood and has several unique physical exam findings to distinguish it from other forms of allergic conjunctivitis. It may involve the tarsal plate, where the papillae enlarge up to 7-8 mm, and are described as having a "cobblestone" appearance, one of the hallmarks of the disease. (*See Figure 1.*) The limbus may reveal yellow-appearing infiltrates and neovascularization. Itching is severe, and photophobia is common. It is not unusual for patients to have repeated episodes of conjunctivitis throughout the year. AKC occurs more commonly in men between the ages of 30 and 50. As opposed to VKC, the lower lid is more commonly involved.[23] These more severe types of ocular allergy are more complicated to treat than simple allergic conjunctivitis (SAC and PAC) and usually involve the use of topical corticosteroids under the supervision of an ophthalmologist.

Giant Cell Papillary Conjunctivitis. GPC is commonly classified with other allergic entities because it involves mast cell degranulation and elicits a type 1 hypersensitivity reaction. However, it differs in its immunologic features that are not truly typical of an allergic reaction. GPC is a condition caused by chronic micro-trauma, most commonly seen in contact lens wearers.[26] Patients with GPC present with typical conjunctivitis symptoms. The examination reveals large papillae on the upper tarsal palpebrae and a stringy mucus discharge. There is no mortality with this disease, and treatment, although variable, generally yields favorable results. Temporarily stopping contact lens use is essential until GPC resolves. Topical combination antihistamine/mast cell stabilizers, nonsteroidal anti-inflammatory drugs (NSAIDs), and occasionally corticosteroids are used. A subset of patients will never tolerate contact lenses due to this entity.

Diagnosis of allergic conjunctivitis in the ED is based on the history and clinical presentation. Treatment of allergic conjunctivitis depends on the cause, but usually involves topical agents that have a rapid onset of action. Studies have shown that topical ocular treatment may be equal to nasal or oral agents for the ocular component of simple allergic conjunctivitis.[27,28] Oral antihistamines are helpful, however, in cases of allergic rhinoconjunctivitis to relieve the nasal symptoms that accompany the ocular allergy. Popular treatments are the antihistamines and combination drops that have two or more antihistamine/mast cell stabilizer/anti-inflammatory components. Vasoconstrictive agents, mast cell inhibitors, NSAIDs, and, in complicated cases, corticosteroids, may be used alone or in combination. Topical vasoconstrictors may cause a rebound effect or a hypersensitivity reaction that should be taken into account when prescribing this medication, and should not be used as first-line agents. Table 1 provides a list of some common topical medications used for the various types of allergic conjunctivitis. Avoidance of known allergens is important when counseling these patients.

Chemical Conjunctivitis. The normal pH of the eye is approximately 7.0-7.4. Acids and alkali can seriously damage the conjunctiva and deeper structures of the eye. Ocular burns occur in up to 18% of patients who present with eye trauma[29]; the majority of these are from chemical burns. Chemical burns can denature and coagulate eye proteins and cause ischemia to vascular structures. Patients with chemical exposure to the eye should undergo immediate copious irrigation prior to a complete history and physical examination. The determining factor in morbidity from acid or alkali burns to the eye is the duration of exposure and the pH of the offending agent. Alkali burns are lipophilic and penetrate the eye quickly, causing deep necrosis, a condition termed "liquefactive" necrosis. If the pH is higher than 12, irreversible damage will likely occur.

Acids produce a coagulation necrosis. They denature proteins, causing eschar formation that functions as a barrier to further acid penetration, and a resultant superficial burn. The exception to the superficial nature of acid burns is hydrofluoric acid, which is a weak acid with rapid penetration into the cornea, causing a liquefactive necrosis injury. Treatment of acid or alkali burns in the ED involves prompt and copious irrigation to return the eye to a pH of about 7.4. A topical anesthetic applied prior to irrigation helps in pain management. A Morgan lens can be used to deliver low-pressure irrigation using 1 to 2 liters of irrigation solution (tap water is acceptable) over 30 to 60 minutes, at which time the pH is checked using litmus paper. The pH should be rechecked 30 to 60 minutes after an optimal pH is reached,

Table 1: Common Topical Medications for Allergic Conjunctivitis

Antihistamines	Mast Cell Stabilizers	NSAIDs	Antihistamine/ Mast Cell Stabilizer Combination	Corticosteroids
Selective histamine (H1) receptor antagonists	Stops the release of inflammatory mediators from the mast cell	Inhibit the production of prostaglandins and thromboxane	See the individual properties of each. In addition, some have anti-inflammatory properties.	Work intracellularly to inhibit production of pro-inflammatory mediators
Antazoline 0.05%	Alamast 0.1%	Ketorolac 0.5%	Olopatadine 0.01%	Loteprednol etabonate 0.5%
Azelastine 0.05%	Opticrom 4%	Diclofenac 0.1%	Ketotifen 0.025%	Prednisolone acetate 1.0%

and irrigation restarted if necessary. Ophthalmology should be consulted if there is significant injury to the cornea or sclera.

Corneal Abrasion and Corneal Ulcer

Keratitis refers to inflammation of the cornea and includes both corneal abrasion and corneal ulcer. Corneal abrasions are scratches to the superficial layers of the cornea and usually have a good prognosis with no visual loss. When injury or inflammation involve the deeper layers of the cornea, it is termed corneal ulcer, a more serious process that may cause scar formation and resultant visual loss if disruption occurs in the visual field. In severe cases, corneal perforation may occur. In areas of the developing world, corneal ulcers are a major cause of blindness.[30] Predisposition for both of these processes occurs in individuals with conditions that cause dry eyes or decreased corneal sensation. Examples of this include patients with Bell's palsy and incomplete eyelid closure, or patients with corneal viral infections or chemical burns causing decreased corneal sensation.

Infection is the leading cause of corneal ulceration, and often complicates non-infectious etiologies as well. Bacteria, viruses, fungi, and amoeba are common causes and, with the exception of viruses, usually invade a traumatized cornea. In some cases, the trauma is not apparent, as seen with contact lens use. Bacteria are the most common infectious cause of corneal ulcers. In the United States, the most common bacteria are *Pseudomonas aeruginosa* and *Staphylococcus aureus*.[31] In addition, immune-related conditions such as Lyme disease may localize to the eye as a non-infectious corneal inflammation, or present with ocular complaints as part of a systemic process. Risk factors for corneal ulcer include contact lens use, corticosteroid eye drop use, immunocompromised states, eye trauma, and warm climates.

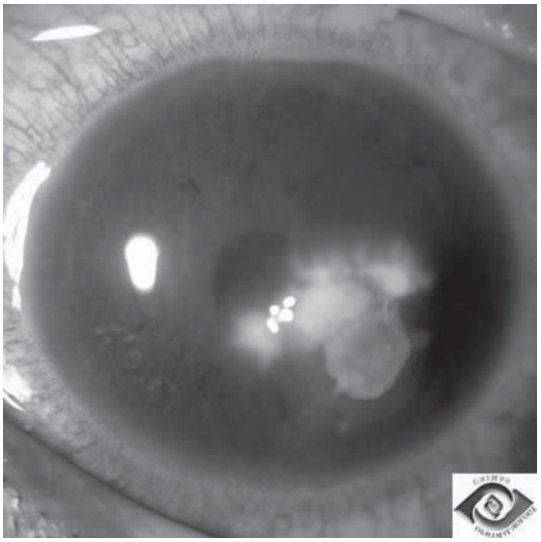
Unlike conjunctivitis, patients typically complain of eye pain and a foreign body sensation. They usually present with blurry vision, photophobia, and excessive tearing. The physical exam reveals these findings, along with conjunctival injection or ciliary flush. Miosis, chemosis, and lid edema may be present. The physical exam must include upper lid eversion to detect and remove any foreign bodies that may be present. A defect in the surface of the cornea can be seen with the slit lamp and confirmed with fluorescence testing. Corneal abrasion and ulcer can be differentiated by the depth and color of the lesion. Corneal abrasions appear clear on slit lamp examination. In corneal ulcer, opacification of the cornea occurs and appears as a white opacity often seen on simple visual inspection. (*See Figure 2.*) Slit lamp confirms the finding. If herpetic keratitis is present, classic dendrite formation is seen with florescence application.

The physician may encounter several distinct types of keratitis:

- UV keratitis occurs from exposure to natural or artificial ultraviolet light. It is seen in welders, people with exposure to bright sunlight or reflected light off snow or sand, and in patients using tanning salons without protective eyewear. Symptoms are usually delayed for hours after exposure. It causes a painful punctuate keratitis, often on the inferior portion of the cornea, which appears as small pinpoint defects on fluorescein examination.
- Herpes keratitis may occur with HSV (usually type 1) or varicella-zoster virus. It is recognized by a classic dendritic pattern on fluorescein staining of the eye.
- Contact lens acute red eye (CLARE) is caused by protein or bacteria deposits on the contact lens that inflame the cornea. It is more common in extended-use lenses. Gram-negative bacteria (Pseudomonas, Serratia, and *Haemophilus influenzae*) are isolated.
- Diffuse lamellar keratitis is a complication of Lasik surgery seen most commonly the day after surgery, but can occur within the first week. Sterile infiltrates elicit an inflammatory reaction in the cornea around the surgical incision site. Because it may be difficult for the ED physician to distinguish this entity from infectious keratitis, an ophthalmologist should be involved in the care of these patients.

Because the etiology is often not initially known, and to prevent bacterial super infection, topical

Figure 2: Corneal Ulcer Appearing as a White Opacification on the Surface of the Eye



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antibiotics are usually given. Pain may be treated with oral medication. In corneal ulcers, topical NSAIDs have been used with good results.[32] Cycloplegics may alleviate ciliary spasm. For viral keratitis, topical or oral antivirals such as acyclovir, and topical or oral steroids should be considered. Fungal keratitis is a contraindication to topical corticosteroid use. Ophthalmology should be consulted when a corneal ulcer is present and topical steroids are prescribed.

Scleritis/Episcleritis

Inflammation of the sclera may occur in the more superficial episclera, which lies just beneath the conjunctiva, or in the adjacent inner layer of the sclera. These two processes are termed episcleritis and scleritis, respectively, and differ significantly in severity and potential for visual loss. They are relatively uncommon disorders.

Episcleritis is a benign disease affecting the external portion of the eye and the superficial vascular plexus of the sclera. Its exact pathogenesis is unknown. Like other forms of inflammatory eye disease, it has been linked to certain diseases, such as systemic lupus, rheumatoid arthritis, and inflammatory bowel disease,[33-35] which occur in a minority of patients. The majority of cases occur in young to middle-aged females.[36] Episcleritis has two clinical presentations: simple and nodular. Usually episcleral hyperemia is localized, often with inflammation of the overlying bulbar conjunctiva. Nodular episcleritis presents with a focal nodule surrounded by hyperemia. Unlike simple episcleritis, which usually resolves spontaneously in several weeks, nodular episcleritis is slower to clear and is more likely to have recurrence. Both can present with acute, focal, or generalized hyperemia and mild eye discomfort. Symptoms may be bilateral, and visual acuity is unaffected. It is differentiated from conjunctivitis by its focality and lack of discharge, although tearing is common. On slit lamp examination, there is episcleral vascular congestion and edema of the surrounding episcleral tissue. A localized whitish-appearing nodule may be seen within the area of hyperemia. Uveitis and keratitis may complicate a minority of cases. Treatment is usually supportive. Topical corticosteroids, on a limited basis, may be prescribed to decrease inflammation in the extremely symptomatic patient. They are effective in treating episcleritis, but the risk of their use should be considered in treating this relatively benign disease. Topical NSAIDs have been suggested, but are of questionable efficacy.[37] Oral NSAIDs may also be used. This condition may be recurrent, but does not progress to scleritis or cause visual loss.

Scleritis. Scleritis a more serious disease and should be approached by the ED physician as an ocular emergency. An immune-complex vasculitis-type reaction may account for the pathogenesis of this disease.[38] In up to 50% of patients, it occurs in conjunction with an autoimmune systemic inflammatory disease.[39] Rheumatoid arthritis and Wegener's granulomatosis are the most frequently associated autoimmune diseases.[40] In a minority of patients, it is caused by infection, as a complication from ocular surgery, a medication reaction, or secondary to malignancy. Scleritis involves the anterior or posterior portion of the eye. When anterior, the inflamed scleral structures are visible on exam, but when the disease is posterior, the ocular changes are hidden from view.

There are five clinically recognized subtypes in this disease: four anterior and one posterior. Approximately 90% of cases are of the anterior type.[41] Anterior scleritis is either diffuse, nodular, necrotizing with inflammation, or necrotizing without inflammation (scleromalacia perforans). The most common subtypes are anterior diffuse and anterior nodular. Anterior necrotizing scleritis with inflammation is not common, but is the most severe form of the disease and the most likely to result in loss of vision. Posterior scleritis occurs in 2-12% of cases.[42] The patient usually presents with a normally appearing painful eye and, therefore, the diagnosis is often missed. Eye pain and a decrease in visual acuity should prompt the clinician to consider this diagnosis. It is associated with anterior scleritis in over 50% of cases, at which point sclera inflammation is visible. Complications include uveitis, retinal detachment, retinal hemorrhage, macular edema, cataracts, and

Figure 3: Keratic Precipitates Seen on the Cornea in Anterior Uveitis

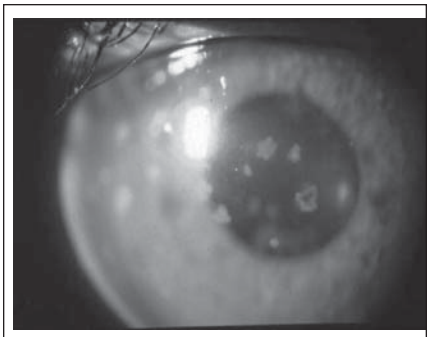
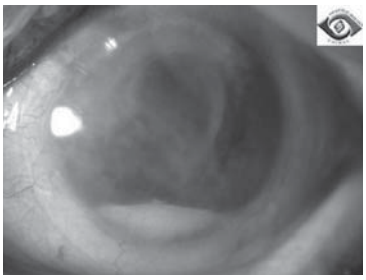


Figure 4: Classic Appearance of a Hypopyon in the Anterior Chamber



ophthalmoplegia.

Aside from sclera vascular injection, the prominent symptom in scleritis is pain, which may be orbital or periorbital. Patients typically present with gradual onset of symptoms. Reduced visual acuity may be present. Examination is variable depending on the subtype but classically reveals tenderness to palpation, and focal or diffuse hyperemia, which is described as having a violaceous hue due to the depth of the involved blood vessels. Proptosis and lid edema may be present. There may be an associated underlying systemic disorder, which should increase the clinician's suspicion for this diagnosis.

It can be difficult to differentiate episcleritis from scleritis. On exam, episcleral vessels will move when prodded with a cotton-tipped swab. Also, if you instill a 10% solution of pilocarpine, episcleral vessels will blanch, while deeper sclera vessels will not. Imaging modalities useful in diagnosing scleritis include b-mode ultrasonography and CT scanning,[40] which are helpful in elucidating disease at the back of the eye. Treatment of non-infectious scleritis is always systemic. Non-necrotizing scleritis is treated with oral NSAIDs, oral steroids, or a combination of both. Peri-oribital or subconjunctival steroid injections have been used if necrotizing disease is present but are beyond the scope of ED practice. In necrotizing scleritis, oral steroids, immunosuppressants such as cyclophosphamide or cyclosporine, or a combination of both is used. In posterior scleritis, the underlying cause must be treated along with oral NSAIDs, steroids, or both. In infectious scleritis, antimicrobial treatment is given systemically with or without topical antimicrobials. Because of the high incidence of associated systemic disease, patients with scleritis should be referred to a primary practitioner for a thorough medical evaluation.

Uveitis/Iritis

The uvea is the portion of the eye that includes the iris and the ciliary body anteriorly and the choroid posteriorly. Inflammation of the anterior uveal tract may involve the iris, a condition known as uveitis or iritis. If it involves the iris and ciliary body, it is called iridocyclitis. When posterior uveal structures are involved in inflammatory processes, the condition has multiple terms, including pars planitis, intermediate uveitis, retinitis, or chorioretinitis. Panuveitis is inflammation of all uveal structures. The pathogenesis of uveitis is unknown, but it is strongly associated with a variety of systemic disorders. Causes include infectious, idiopathic, and localized or systemic autoimmune disorders. In addition, the term "masquerade syndrome" is used to refer to disorders, often malignant, that cause similar symptoms to uveitis, although they do not represent the true disease. Occasionally, trauma and drugs may also cause this disease. Factors in the history that should heighten the clinician's suspicion for uveitis include a past or current history of autoimmune disorders, HIV disease, recent ocular surgery, or diseases such as syphilis, tuberculosis, toxoplasmosis, sarcoidosis, and Lyme disease.

Uveitis may be classified as anterior or posterior, or by clinical course as acute, recurrent, or chronic.[44] In the Western world, the most common form of uveitis is acute, anterior, nongranulomatous disease associated with HLA-B27 positive patients.[45,46] In patients who have symptomatic systemic disease, most are male with a spondyloarthropathy such as ankylosing spondylitis or reactive arthritis.

Anterior uveitis commonly presents with pain that occurs over hours to days, ciliary flush, photophobia, miosis, and variable visual loss. Posterior uveitis patients do not typically have a red eye or pain but variable degrees of visual loss or the perception of "floaters" in the visual field. Slit lamp exam may show white cell deposits on the cornea, termed "keratic precipitates." (*See Figure 3.*) The hallmark of uveitis is flare and cells in the anterior chamber on slit lamp exam. Inflammatory white blood cells are directly visualized, while the "flare," a foggy haze, results from proteinaceous material leaking out of inflamed vasculature into the anterior chamber. If white cells settle in a meniscus-like fashion in the anterior chamber, it is termed a hypopyon. (*See Figure 4.*) Posterior uveitis requires specialized equipment to visualize the back of the eye, which is out of the scope of the ED physician's practice.

The goal in managing anterior uveitis is to relieve pain and inflammation and to prevent possible complications such as glaucoma, synechiae formation, and visual loss. The mainstay in treatment of non-infectious uveitis is topical corticosteroids, often with a topical mydriatic agent, such as atropine, that helps relieve spasm and prevent posterior

Table 2: Common Medications in Acute Angle Closure Glaucoma

Class of Medication	Mode of Action	Representative Drug	Initial Dose
Alpha-adrenergic agonists	↓ AH production ↓ Resistance to aqueous outflow	Apraclonidine	1 drop of 0.5 or 1%
Beta blockers	↓ AH production	Timolol maleate	1 drop of 0.25 or 0.5%
Parasympathomimetics	Contracts ciliary muscle: miosis ↑ Outflow of AH	Pilocarpine	1-2 drops
Prostaglandin analogs	↑ Outflow of AH	Latanoprost	
Carbonic anhydrase inhibitors	↓ AH production	Acetazolamide	500 mg IV, then 500 mg PO
Steroids	↓ Intraocular inflammation	Prednisolone	1-2 drops
Hyperosmotic medication	Osmotic diuresis ↓ Vitreous volume	Mannitol Isosorbide	1.5-2 g/kg IV (20% solution) over 30 minutes 1.5 g/kg PO
Antiemetic	↓ Nausea	Ondansetron	4 mg IV or PO
Analgesic	↓ Pain	NSAID, narcotic	

Note: Adult doses are listed; AH = aqueous humor

synechiae from forming. If topical steroid treatment fails, systemic or peri-ocular steroids can be given. When posterior uveitis is diagnosed, systemic corticosteroids are used. Recent developments in treatment of posterior uveitis have focused on local, intraocular corticosteroids and surgically implanted devices that deliver steroids in a sustained-release manner.[47] In infectious uveitis, the underlying cause is treated. The main causes of visual loss are from macular edema, glaucoma, and cataract formation, which are usually seen in patients with chronic disease.

Acute Angle Closure Glaucoma

Acute angle closure glaucoma (AACG) is a true ocular emergency that requires emergent ophthalmology consultation in the ED. It occurs when there is a sudden, rapid increase in intraocular pressure due to closure of the anterior chamber angle. Under normal circumstances, aqueous humor drains out of the anterior chamber of the eye via the trabecular meshwork located at the angle of the anterior chamber in Schlemm's canal. Anything that blocks this angle will prevent drainage of aqueous humor and increase the normal pressure inside the eye. There are two main types of AACG: Primary angle closure and secondary angle closure. In primary angle closure, patients have anatomic abnormalities that account for angle narrowing or closure. In secondary angle closure, a secondary process such as hemorrhage or tumor is responsible for mechanically deforming the structures at the angle of the anterior chamber to cause its closure. Overall, the most common cause of obstruction occurs when the peripheral iris is pushed up against the angle and becomes a mechanical barrier to normal drainage. In this situation, physical contact between the iris and lens occurs, which is termed "pupillary block."

The highest rates are seen among patients of Eskimo or Asian decent.[48] Risk factors include farsighted individuals who have inherently shallow anterior chambers and narrow anterior chamber angles. Because hyperopia (farsightedness) occurs with advancing age, it is more common in the elderly. In addition, with advanced age comes enlargement of the lens, which pushes the iris forward and predisposes individuals to this disease. Other risk factors include a family history of AACG. Medications, used topically or systemically, can also contribute to AAGC. Adrenergic agents, anticholinergic and cholinergic medication, and sulfa-based drugs have been implicated.[49] The mechanisms in drug-induced AACG are pupillary dilatation, anterior displacement of the lens-iris diaphragm, and ciliary body edema.[50] Moving from a dark environment to a light one, which is classically described as coming out of a dark movie theatre into bright light, can acutely dilate the pupil and precipitate an attack. Although unusual, bilateral AACG can occur and is often attributed to underlying systemic disease.[51-53]

Symptoms of AACG include eye pain, headache, nausea, blurred vision, tearing, and the appearance of halos around lights (a result of corneal edema). Abdominal pain, nausea, and vomiting have also been reported. The exam may reveal ciliary flush, generalized conjunctival hyperemia, a fixed or sluggish mid-dilated pupil, corneal edema, and a shallow anterior chamber. Visual acuity is diminished and intraocular pressure is elevated. In normal eyes, intraocular pressure ranges 8-21 mmHg. When an eye suffers from acute angle closure,

the pressure often rises above 30 mmHg. Prophylactic treatment is often initiated in the unaffected eye if a narrow angle is seen on slit lamp examination. Rarely, the process resolves spontaneously, but recurrences are common. If untreated, optic neuropathy rapidly ensues and permanent vision loss may occur in hours to days. Emergent treatment, therefore, is aimed at initially lowering the intraocular pressure to below 40 mmHg as quickly as possible. Examples of common medications to reduce intraocular pressure, reverse angle closure, and treat the systemic symptoms of AACG are listed in Table 2. A common approach is to give topical beta-blockers, alpha-adrenergic blockers, or prostaglandin inhibitors and oral or intravenous acetazolamide to initially decrease intraocular pressure. Pressure should be rechecked one hour later. Once intraocular pressure is lowered, pilocarpine is given to vasoconstrict the pupil in hopes of widening the anterior chamber angle. If pilocarpine is given before intraocular pressure is reduced, it is generally ineffective because elevated intraocular pressures cause paralysis of the iris. Medication to dilate the eye must be avoided, as this will exacerbate AACG. If these modalities are unsuccessful in decreasing intraocular pressure, laser peripheral iridotomy is performed by an experienced ophthalmologist.

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Physician CME Questions

11. Which of the following is true of episcleritis?
    A. It is best treated with systemic steroids.
    B. It is more common in children.
    C. It causes extreme pain.
    D. It is often self-limiting and resolves without treatment.

12. Which of the following are true concerning acute angle closure glaucoma?
    A. Eye pressures often exceed 30 mmHg.
    B. Emergent treatment is aimed at lowering intraocular pressure and reversing angle closure.
    C. It requires emergent ophthalmology consultation.
    D. All of the above

13. The initial management of alkali burns to the eye is:
    A. topical steroid drops
    B. dilute topical acid solution to counteract the effect of the base
    C. copious irrigation to return pH to normal
    D. systemic analgesia and oral antibiotics

14. Preauricular lymphadenopathy is most often found in which of the following?
    A. uveitis
    B. adenovirus conjunctivitis
    C. episcleritis
    D. bacterial conjunctivitis

15. Which of the following statements regarding acute uveitis is *not true*?
    A. Pain and photophobia are common.
    B. It may involve inflammation of the iris, ciliary body, or choroid.
    C. It is associated with contact lens use.
    D. Anterior uveitis is more common than posterior uveitis.

16. Which of the following is the most common cause of conjunctivitis in the newborn?
    A. *Chlamydia trachomatis*
    B. Staphylococcus species
    C. *E. coli*
    D. Adenovirus

17. Ocular pain, ciliary flush, corneal edema, and a fixed mid-dilated pupil are classic symptoms of:
    A. iritis
    B. conjunctivitis
    C. acute angle closure glaucoma
    D. keratoconjunctivitis

18. Large, "cobblestone" appearing papillae on the tarsal plate are seen in:
    A. Neisseria conjunctivitis
    B. herpetic keratitis
    C. vernal keratoconjunctivitis
    D. scleritis

19. Which two autoimmune diseases are most frequently associated with scleritis?
    A. rheumatoid arthritis and Wegener's granulomatosis
    B. antiphospholipid syndrome and atopic dermatitis
    C. Behçet's syndrome and celiac disease
    D. Cushing's syndrome and erythema nodosum

20. All of the following are more indicative episcleritis than scleritis *except*:
    A. benign disease
    B. it can present with eye pain and decreased visual acuity
    C. hyperemic vessels blanch with topical instillation of pilocarpine
    D. lack of significant pain

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Executive Editor: Shelly Morrow Mark

Managing Editor: Leslie Hamlin

GST Registration No.: R128870672

Periodicals Postage Paid at Atlanta, GA 30304 and at additional mailing offices.

POSTMASTER: Send address changes to Emergency Medicine Reports, P.O. Box 105109, Atlanta, GA 30348.

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The Red Eye

Common Topical Medications for Allergic Conjunctivitis

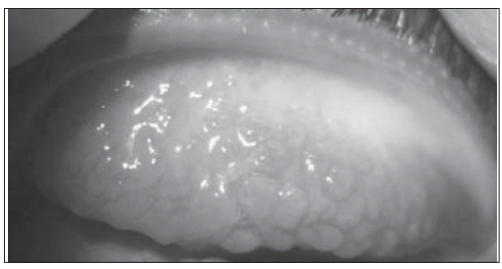
Antihistamines	Mast Cell Stabilizers	NSAIDs	Antihistamine/ Mast Cell Stabilizer Combination	Corticosteroids
Selective histamine (H1) receptor antagonists	Stops the release of inflammatory mediators from the mast cell	Inhibit the production of prostaglandins and thromboxane	See the individual properties of each. In addition, some have anti-inflammatory properties.	Work intracellularly to inhibit production of pro-inflammatory mediators
Antazoline 0.05%	Alamast 0.1%	Ketorolac 0.5%	Olopatadine 0.01%	Loteprednol etabonate 0.5%
Azelastine 0.05%	Opticrom 4%	Diclofenac 0.1%	Ketotifen 0.025%	Prednisolone acetate 1.0%

Common Medications in Acute Angle Closure Glaucoma

Class of Medication	Mode of Action	Representative Drug	Initial Dose
Alpha-adrenergic agonists	↓ AH production ↓ Resistance to aqueous outflow	Apraclonidine	1 drop of 0.5 or 1%
Beta blockers	↓ AH production	Timolol maleate	1 drop of 0.25 or 0.5%
Parasympathomimetics	Contracts ciliary muscle: miosis ↑ Outflow of AH	Pilocarpine	1-2 drops
Prostaglandin analogs	↑ Outflow of AH	Latanoprost	
Carbonic anhydrase inhibitors	↓ AH production	Acetazolamide	500 mg IV, then 500 mg PO
Steroids	↓ Intraocular inflammation	Prednisolone	1-2 drops
Hyperosmotic medication	Osmotic diuresis ↓ Vitreous volume	Mannitol Isosorbide	1.5-2 g/kg IV (20% solution) over 30 minutes 1.5 g/kg PO
Antiemetic	↓ Nausea	Ondansetron	4 mg IV or PO
Analgesic	↓ Pain	NSAID, narcotic	

Note: Adult doses are listed; AH = aqueous humor

Large Papillae with a Classic "Cobblestone" Appearance Seen in Vernal Keratoconjunctivitis



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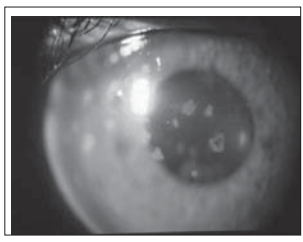
Keratic Precipitates Seen on the Cornea in Anterior Uveitis



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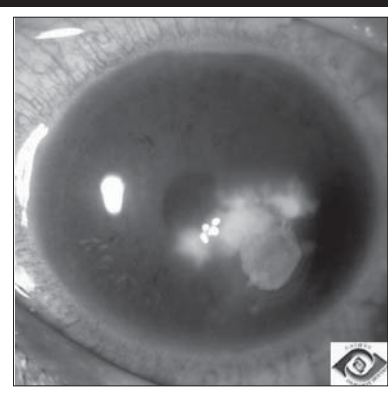
Corneal Ulcer Appearing as a White Opacification on the Surface of the Eye



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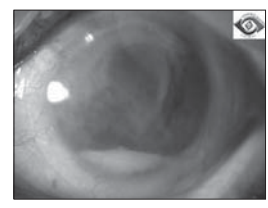
Classic Appearance of a Hypopyon in the Anterior Chamber



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Supplement to *Emergency Medicine Reports*, July 4, 2011: "The Red Eye." *Author:* **Pamela Arsove, MD, FACEP**, Associate Residency Director, Department of Emergency Medicine, Long Island Jewish Medical Center, New Hyde Park, NY; Assistant Professor of Emergency Medicine, Hofstra North Short – Long Island Jewish School of Medicine.

Trauma Reports

EVIDENCE-BASED MEDICINE FOR THE ED

Volume 12, Number 4 Jul/Aug 2011

See new CME/CNE testing procedures on page 11.

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Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Dietrich (editor in chief), Drs. Bond and Cobert (authors), Dr. Perron (peer reviewer), and Ms. Behrens (nurse reviewer) report no relationships with companies related to this field of study. Ms. Mark (executive editor), and Ms. Hamlin (managing editor) report no relationships with companies related to the field of study covered by this CME activity.

AHC Media

Knee and Patellar Dislocations: Identifying Subtleties for Optimal Recognition and Management

Knee dislocations have the potential to result in significant morbidity and mortality if not correctly diagnosed and optimally managed. Early identification and treatment of neurovascular injury and compartment syndrome may avert disaster for the patient. Recognition of hard signs of vascular injury mandate immediate surgical exploration, whereas soft signs of vascular injury require further diagnostic evaluation. Closed reduction may be performed when emergency surgery is not indicated. Following reduction, hospital admission is recommended to monitor for neurovascular compromise.

Patellar dislocations are common, and the vast majority are managed by the ED physician. Acute surgical management is uncommon and indicated for persistent patellar subluxation and detachment of the vastus medialis obliquus muscle and medical retinaculum from the medial aspect of the patella.

The authors present a comprehensive and detailed review of the diagnosis and management of knee and patellar dislocations. Common pitfalls and diagnostic advances are reviewed and discussed to maximize patient outcome and decrease long-term functional morbidity.

—The Editor

Introduction: Definition and Classification

A knee dislocation is a complete disruption of the articulation between the tibia and femur. It is often the result of high-energy impacts from sports and motor vehicle collisions. Traumatic dislocations of the knee, while uncommon (with an incidence of 0.02%), can result in significant morbidity and mortality.[1] Young adult males are more frequently the victims of these injuries, fitting the epidemiologic profile of trauma patients in general. The incidence of knee dislocations is probably significantly underreported because many dislocations reduce spontaneously before the affected person reaches a medical facility, so the true nature of the injury is never realized. If the dislocation does not reduce spontaneously, the condition is classified as a "locked" dislocation, which carries significant risk of long-term functional, neurologic, and vascular injury or dysfunction. Therefore, prompt evaluation, stabilization, diagnostic assessment, and treatment are imperative to achieve the best functional outcome.

Multiple classification systems for knee dislocations have been devised, based on the force of the mechanism of injury (high, low, ultra-low), its acute versus chronic nature, or the number of ligaments injured. The widely accepted Schenck's classification system of knee dislocation (*see Table 1*), based on the number of ligaments torn, is designed for operative planning and comparison with similar injuries.[2] In Type I dislocations, one cruciate ligament and one collateral ligament are torn; the second cruciate and collateral ligaments remain

Executive Summary

- The most common clinical classification system for knee dislocations is based on the position of the tibia in relation to the femur. Thus, the dislocation can be classified as anterior (most common), posterior, lateral, medial, or rotary (very rare).
- About 40% of people with knee dislocations have an associated popliteal artery injury.
- Damage to the peroneal nerve has been reported in 25% to 40% of patients with knee dislocations and is more common in those with lateral and posterolateral dislocations.
- Two studies have shown that a normal ABI has an NPV of 100%. However, since intimal tears or dissections can present with normal ABIs initially but change as a thrombus forms and occludes the popliteal artery, patients with knee dislocations need to be admitted for serial examinations.

intact. In Type II dislocations, both cruciate ligaments are torn and the collateral ligaments remain intact. Type III dislocations are subdivided into M and L, based on whether the medial or lateral collateral ligament is torn. In Type III dislocations, both cruciate ligaments and one collateral ligament are torn. In Type IV dislocations, all four ligaments (both cruciate and both collateral ligaments) are torn and the posterolateral corner (PLC) is injured. In Type V dislocations, a periarticular fracture is also present.

The most common clinical classification system is based on the position of the tibia in relation to the femur. Thus, the dislocation can be classified as anterior (most common), posterior, lateral, medial, or rotary (very rare).[3] Combinations can also occur, with the most common being posterolateral.[4] This classification system is of limited value for dislocations that reduce spontaneously prior to medical evaluation. In these cases, the Schenck classification system should be used.

Table 1. Schenck Classification System for Knee Dislocations

I	Single cruciate ligament plus a collateral ligament	ACL or PCL plus a collateral
II	ACL/PCL	Collaterals intact
IIIM	ACL/PCL/MCL	LCL intact
IIIL	ACL/PCL/LCL	MCL intact
IV	ACL/PCL/MCL/LCL and PLC	
V	Fracture Dislocation	

ACL = Anterior cruciate ligament, PCL = Posterior cruciate ligament, MCL = medial cruciate ligament, LCL = lateral collateral ligament, PLC = posterolateral corner

Anatomy and Pathophysiology

The knee joint consists of three articulations between the femur, tibia, and fibula: the tibiofemoral, patellofemoral, and proximal tibiofibular articulations. The patella, the largest sesamoid bone in the body, sits directly anterior to the joint. The patella enhances and aligns the force of the quadriceps muscle during knee extension and keeps the quadriceps force from being pulled laterally. The strong medial patellofemoral ligament (MPFL) stabilizes the patella and offsets the lateralizing force of the quadriceps muscle.

Four ligaments stabilize the knee joint. The anterior cruciate ligament (ACL) prevents forward movement of the tibia in relation to the femur, while the posterior cruciate ligament (PCL) prevents posterior movement of the tibia. The lateral collateral ligament (LCL) stabilizes the knee laterally and is the primary restraint to varus angulation, while resisting internal rotation forces. Finally, the medial collateral ligament (MCL) provides medial stability and is the primary restraint to valgus angulation. The knee also has two menisci (medial and lateral), which are fibrocartilaginous structures that act as shock absorbers between the knee and femur, reducing friction and decreasing the contact area between the bones.

The posterolateral corner of the knee is critical to its stability, particularly in the varus and rotatory planes. The anatomy of this region is formed by the deep, middle, and superficial layers of tissue. The deep layer contributes most of the stability to this region and is composed of the popliteus muscle, the popliteofibular ligament, the LCL, the arcuate ligament, and the fabellofibular ligament.[5] The peroneal nerve travels through the deep layer. The middle layer consists of the patellar retinaculum and the patellofemoral ligament. The superficial layer consists of the lateral fascia, the iliotibial tract, and the tendon of the biceps femoris muscle. Isolated posterolateral corner

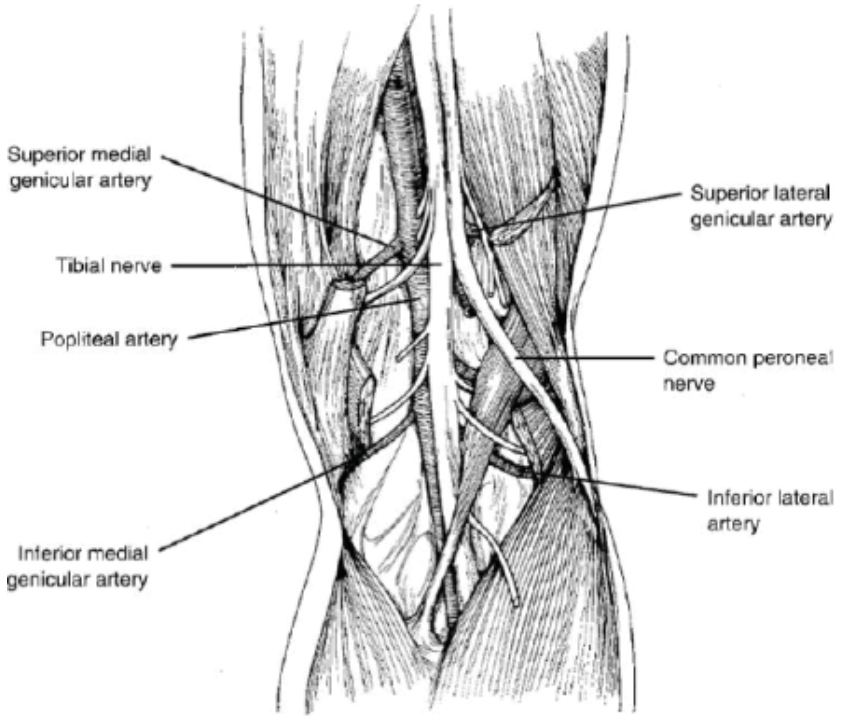
injuries are rare; this region is commonly injured in conjunction with ACL or PCL tears.[6]

The popliteal artery provides the major arterial supply to the lower leg. It is tethered proximally by the adductor hiatus of the thigh and distally by the proximal soleus fascial arch. (*See Figure 1.*) The popliteal artery is susceptible to injury during dislocation by direct blow, stretch, or shearing forces. A wide range of injuries can occur to the popliteal artery (e.g., intimal flap tear, thrombosis, pseudoaneurysm, and transection). The knee has poor collateral circulation from the genicular arteries, which may be easily avulsed or thrombosed during a dislocation. As a result, damage to the popliteal artery can lead to significant morbidity.

The sciatic nerve divides into the tibial and common peroneal nerves at the distal femur. The common peroneal nerve divides into the sural cutaneous, deep peroneal, and superficial peroneal nerves as it wraps around the head of the fibula. Knee dislocations can injure any of these nerves. The peroneal nerve is at high risk for injury during knee dislocations because it has a superficial course through the popliteal fossa and runs very close to the fibular neck. The common peroneal nerve provides motor innervation to the short head of the biceps femoris. Its branch, the lateral sural cutaneous nerve, provides sensory innervation to the superior lateral side of the leg. The superficial peroneal nerve provides motor innervation to the lateral compartment of the leg and sensory innervation to the lateral aspect of the lower leg and the dorsum of the foot. The deep peroneal nerve provides motor innervation to the anterior compartment of the leg and sensory innervation to the first dorsal web space.

The tibial nerve is also at risk for injury during knee dislocations. It runs medially to the common peroneal nerve in the popliteal fossa and extends straight down distally to innervate the posterior compartment of the lower leg and the plantar aspect of the foot.

Figure 1. Anatomy of the Popliteal Space



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Knee Dislocation

A knee dislocation (*Figure 2*) is defined as a complete disruption of the joint such that the articular surfaces are no longer in contact; dislocations also include knees with multi-ligamentous injury or multi-directional instability.[7] Subluxation is similar to a dislocation in that the joint is disrupted; however, the articular surfaces remain partially intact. Knee dislocations may induce multiple ligamentous injuries, but it is possible to have a dislocation without bicruciate ligament tears. Knee dislocations are classified as anterior, posterior, medial, lateral, rotary, or a combination of these directions, which are based on the tibia's position in relation to the femur. Anterior dislocations are the most common classification, with an incidence of 31%.[8] Posterior dislocations often require greater force in the mechanism of injury.

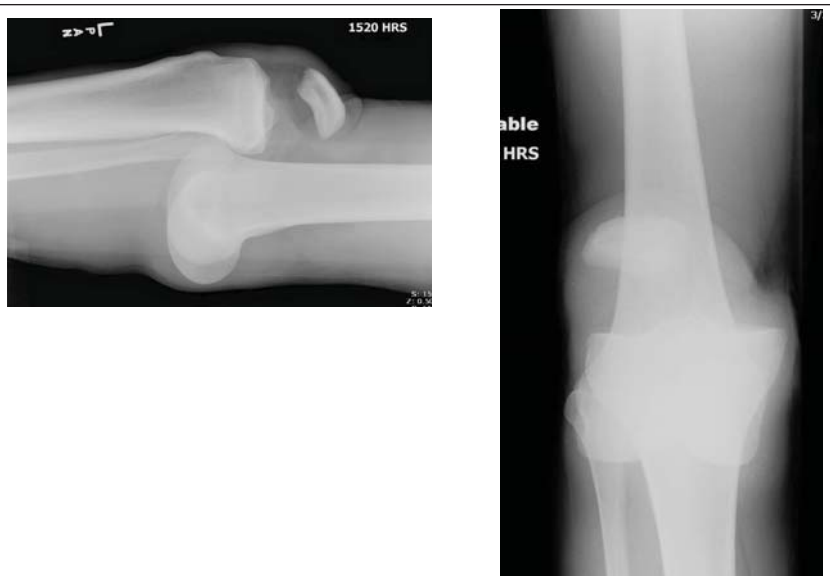
The incidence of fracture among patients with knee dislocation is as high as 40%.[8] Ligamentous injury is variable, depending on the force and direction of the dislocation. Several studies recommend early operative repair of injured ligaments to achieve good functional outcome.[3,9]

About 40% of people with knee dislocations have an associated popliteal artery injury.[8,10] If the pulse is absent, reperfusion must be restored within 6 to 8 hours after injury to minimize the risk of limb necrosis and amputation.[3,9,10] Popliteal artery intimal tears are more common in anterior dislocations; full transection of the artery is more common in posterior dislocations. Because of the poor collateral circulation around the knee, damage to the popliteal artery can result in significant morbidity. In cases of complete ligation of the popliteal artery, necrosis has

been reported in as many as 72.5% of patients.[9]

The reported incidence of neurologic injuries associated with knee dislocations ranges from 16% to 40%.[7] During a dislocation, particularly with forced varus or hyperextension stress, the fibular neck may pull against the peroneal nerve. Damage to the peroneal nerve has been reported in 25% to 40% of patients with knee dislocations and is more common in those with lateral and posterolateral dislocations.[8] The highest risk of injury to the common peroneal nerve (including complete transection) is associated with dislocations combined with disruption of the ACL, PCL, and posterolateral corner.[11] It is not surprising that dislocations resulting from forces high enough to cause multiple ligamentous and bony injuries also result in more severe nerve injuries, including complete transection.

Figure 2. Anterior Knee Dislocation



Images courtesy of Michael C. Bond, MD

Physical Examination

A careful and focused physical examination of the injured extremity must be performed. Gross inspection includes assessment for open injuries and deformity and a skin inspection for mottling and cyanosis. The examiner should compare the injured joint and limb with the uninjured leg. In all three positions, the knee and patella should be inspected and palpated for alignment, deformity, swelling, hemarthrosis, effusion, and open wounds.

Passive and active range of motion should be evaluated. The patient should actively extend the knee from a flexed position. During range of motion, the examiner should feel for crepitus, which may be a sign of cartilage injury. The examiner should also evaluate the patella during active range of motion to look for any abrupt lateral shifting.

The knee must be checked for ligamentous stability. If the history suggests the patient may have had a knee dislocation that reduced spontaneously, the only objective evidence may be the observation of multidirectional knee instability. The patient should be evaluated for anterior or posterior displacement of the tibia when stressed (anterior or posterior drawer signs) and for valgus or varus laxity when the MCL and LCL ligaments are stressed, respectively. In patients who present with a locked dislocation, expedient reduction is important to minimize vessel and nerve strain.

Extremity perfusion and viability are high priorities. The first step in the evaluation for a potential vascular injury includes observation of both hard and soft signs. Hard signs include an expanding hematoma, active hemorrhage or pulsatile bleeding, absent distal pulses, a cold or pale limb, and a palpable thrill or audible bruit.[1,12] The presence of a hard sign is an indication for immediate surgical exploration with vascular reparation. Soft signs of vascular injury include history of hemorrhage, small or non-expanding hematoma, diminished pulses, wound proximity to major vessels, poor skin color, and capillary refill time longer than 2 seconds.[1,12] The presence of a soft sign mandates further diagnostic evaluation aside from the physical examination. Vascular perfusion should be assessed manually by palpation of the dorsalis pedis (DP) and posterior tibial (PT) pulses. A retrospective review by Peck et al found that 7% of patients with blunt lower-extremity trauma had palpable pedal pulses despite significant vascular injury.[13] This observation highlights the limitations of the physical examination to detect vascular injuries. However, in that review, there was no quantification of "palpable pedal pulses." The pulses may have, in fact, been diminished or unequal. Interestingly, a retrospective cohort study showed that no patients with a normal neurovascular examination and no signs of arterial insufficiency or compartment syndrome, in whom angiography was deferred, sustained limb loss or required vascular intervention.[14] Treiman et al found that 4% of patients with a normal pulse examination after a knee dislocation had a popliteal artery injury.[15] None of them required arterial repair. The sensitivity, specificity, and negative predictive value (NPV) of the pedal pulse examination in predicting arterial injury were 85.2%, 93.2%, and 95.3%, respectively. The sensitivity, specificity, and NPV of the pedal pulse examination in detecting a popliteal artery injury requiring operative repair were 100%, 92.4%,

and 100%, respectively.[15] The utility of capillary refill time (CRT) to indicate perfusion has also been scrutinized. CRT has been shown to be an inadequate clinical indicator of vascular perfusion.[16] Skin and subcutaneous tissue can remain viable with much less blood flow than is required by muscle.[10]

An ankle brachial index (ABI) is an inexpensive, noninvasive, quick test that can be done at the bedside to evaluate arterial competency in an injured extremity. Liberal use of arteriography can result in false-positive results, placing the patient at risk for contrast angiography-related complications (e.g., allergic reaction, contrast nephropathy, vessel injury, cost, and delay to diagnosis). During the past 30 years, there has been a significant shift to evaluate potential vascular injuries with soft signs of arterial injury with noninvasive testing first.

Calculation of the ABI begins with the use of Doppler ultrasound to measure the systolic blood pressure (SBP) at the ankle (the DP or PT artery). The SBP of the ankle is divided by the SBP of the ipsilateral (if uninjured) arm.[17] A value greater than 0.9 is normal, and a value less than 0.9 suggests vascular injury. Lynch and Johansen demonstrated that an ABI less than 0.9 had a sensitivity, specificity, and NPV of 87%, 97%, and 99%, respectively, for arterial disruption.[18] Investigators at a trauma center in Turkey found higher sensitivity (95%) and specificity (97%) in patients who had only soft signs of arterial injury.[12] An ABI less than 0.9 with or without soft signs warrants further investigation with arteriography.[19,20] Two studies have shown that a normal ABI has an NPV of 100%.[21,22] In these studies, the authors concluded that emergent angiography is not necessary for patients with a normal ABI. However, since intimal tears or dissections can present with normal ABIs initially but change as a thrombus forms and occludes the popliteal artery, these patients need to be admitted for serial examinations. Preexisting peripheral vascular disease can be a limitation to ABI testing and can result in low ABI values independent of trauma.

Arterial pressure indices (APIs) are an alternative noninvasive measurement. APIs are similar to ABIs except that they are calculated by dividing the distal SBP of the injured extremity by the SBP in the uninjured paired extremity, as measured by Doppler (in other words, comparing one leg with the other).[23] APIs less than 0.9 are considered abnormal and have a diagnostic accuracy up to 95%.[18] Nassoura and colleagues showed that APIs have a sensitivity, specificity, positive predictive value, and negative predictive value of 72.5%, 100%, 100%, and 96%, respectively, for clinically significant injury.[24] Johansen and associates demonstrated that a cutoff ratio of 1.0 for the API increased the negative predictive value to 99%. However, just like ABIs, this measurement can miss intimal flaps, dissections, and false aneurysms, as these injuries do not affect the flow of arterial blood.[20]

In conclusion, the physical examination of patients with knee dislocations has significant limitations in detecting arterial injury, but not in determining the need for operative repair and the risk of limb loss. Patients without indications for emergent surgery who present with abnormal pulses or neurologic deficits should undergo further diagnostic tests (i.e., Doppler ultrasound, angiography, or arteriography).[19] The decision to perform an invasive arterial diagnostic evaluation should be made in consultation with the radiologist, orthopedist, and vascular surgeon.

In a 10-year review by Branco and colleagues, patients with knee dislocations were found to be at high risk for the development of compartment syndrome[25] (more than 40% in patients with combined arterial and venous injuries required fasciotomy). Signs and symptoms of compartment syndrome include painful passive muscle stretch, swelling, pallor, diminished pulses (late sign), and hyperesthesia.[26] Clinical assessment of compartment syndrome may not always be easy because of the high association of neurologic injuries and the patient's resulting altered pain perception. Therefore, it may be necessary to measure intracompartmental pressures directly. Compartment syndrome may not be present initially, so clinical signs and symptoms must be monitored closely during a patient's emergency department (ED) and hospital stay. If markedly increased swelling, increased compartment pressures, or clinical symptoms of compartment syndrome are noted, a four-compartment fasciotomy is needed emergently. It is important for the clinician to remember that compartment syndrome can develop as a result of soft-tissue edema and does not have to be associated with a vascular injury. In the obtunded patient, consider the use of continuous or intermittent invasive compartment pressure measuring devices.

The neurologic status of the limb should be assessed, with close attention to the tibial and common peroneal nerves. Peroneal nerve injury is far more common than other nerve injuries. Findings of peroneal nerve palsy include the inability to dorsiflex the foot ("drop foot") and the presence of sensory deficits on the anterolateral leg and dorsum of the foot. Findings of tibial nerve palsy include the inability to plantar flex the foot and the presence of sensory deficits on the plantar aspect of the foot.

It is imperative that the clinician responsible for the initial neurologic examination document the findings thoroughly. This documentation is especially important for patients who require immediate operative repair and to note the presence of any neural injury that occurred prior to the patient's arrival. A lack of documentation of preoperative neurologic status can have significant medicolegal ramifications if postoperative neurologic deficits are discovered.

Niall et al showed that when the deep peroneal nerve, which supplies the anterior muscle compartment, is injured in association with traumatic

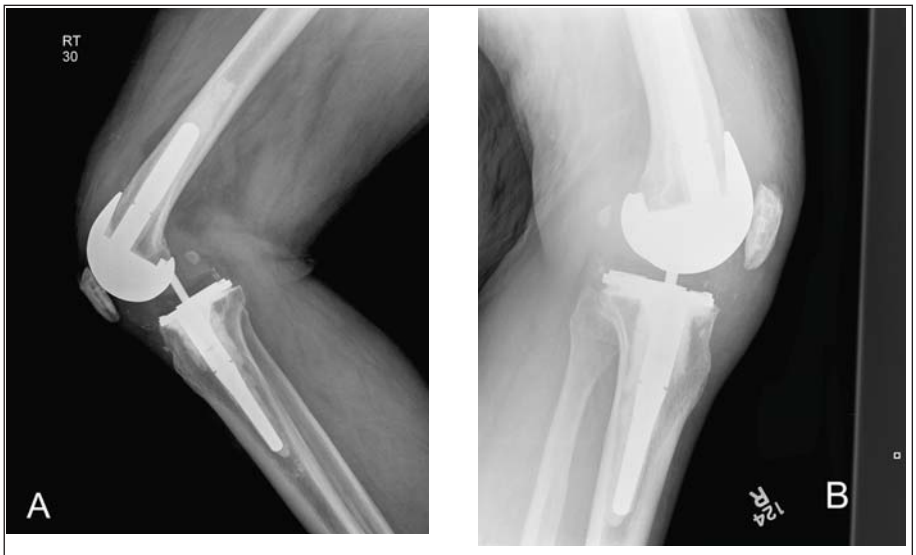
knee dislocation, the prognosis for recovery is worse than in patients in whom the superficial peroneal nerve is injured.[11] In this series, only 21% of patients with injury to the common peroneal nerve achieved complete recovery. Overall, the prognosis for complete neurologic recovery ranges from 14% to 75%, which reflects an uncertain neurologic prognosis.[11]

The ligaments and osteochondral structures should be examined next. The Lachman test is the most sensitive for acute ACL rupture, and the posterior drawer test is most sensitive for acute PCL rupture.[27] MCL and LCL competency is tested with varus and valgus stress, respectively.

Imaging

Radiographic evaluation is an important step in the diagnostic evaluation. Plain radiographs can identify subluxations, dislocations, associated fractures, and effusions. As in most orthopedic evaluations, a joint above and one below the injury should also be imaged to avoid missing associated injuries. Computed tomography (CT) is playing a larger role in the evaluation of knee dislocations because of its higher sensitivity for fractures and associated proximal tibiofibular dislocations.[28] Furthermore, CT angiography, which requires venous cannulation, is safer and has fewer complications than traditional arteriography, which requires arterial cannulation.[29,30] Sufficient evidence exists to suggest that CT angiography may soon surpass arteriography as the gold standard in evaluating arterial injury. Inaba et al report that multi-slice helical CT angiography achieved 100% sensitivity and 100% specificity in diagnosing clinically significant arterial injury.[31] Another study by Soto et al found CT arteriography to have a sensitivity of 95.1% and a specificity of 98.7%.[32] Finally, magnetic resonance imaging (MRI) has higher accuracy than the clinical examination to detect the extent or site of soft-tissue injury (85% to 100% vs. 53% to 82%, respectively).[30] MRI is particularly useful to prepare the patient for surgical repair by an orthopedist, as it can delineate ligamentous and cartilaginous injuries.

Figure 3. Anterior Prosthetic Knee Dislocation



A. Pre-reduction; B. Post-reduction
Images courtesy of Michael C. Bond, MD

Treatment

Initial management of an acute knee dislocation includes recognizing and treating limb-threatening injuries and maximizing long-term joint use. Indications for emergent surgical management include an open wound, compartment syndrome, an irreducible dislocation or failed closed reduction, and a grossly unstable dislocation. Emergent operative vascular repair should be performed when any hard signs of vascular injury are noted. Consider operative repair in patients with evolving soft signs of vascular injury or diminishing ABIs or APIs.

When emergency surgery is not indicated, proceed with closed reduction. Induce procedural sedation in conscious patients, because it allows the reduction to be done with minimal pain and anxiety and it provides a good opportunity to do a thorough ligamentous examination without patient discomfort and voluntary guarding.

Reduction of a knee dislocation requires the aid of an assistant. The assistant should exert firm and constant longitudinal traction on the proximal tibia to ensure that the femur and tibia are distracted. While traction is being held, exert a force on the proximal tibia that is counter to the direction of the dislocation to cause the reduction. For a posterior dislocation, lift the proximal tibia anteriorly to reduce the joint. For an anterior dislocation, push the proximal tibia posteriorly. Avoid the application of pressure on the popliteal space during the reduction attempt, so as to prevent additional stress and traction on the popliteal artery. Many posterolateral dislocations are irreducible with closed reduction attempts because the medial condyle of the femur traps the medial capsule of the joint.[4]

After a closed reduction, recheck the ABIs or APIs. Stabilize the knee with a non-constrictive knee immobilizer. Ideally, the patient should be placed in a hinged knee brace with the knee at 15 degrees of flexion to avoid strain on the popliteal vessels; however, if a hinged brace is not available, a straight-leg knee brace is acceptable. Hospital admission is strongly recommended in light of the high rate of associated

neurovascular injuries and the potential for late manifestations of neurovascular compromise. Serial ABIs and neurologic examinations should be performed hourly for up to 48 hours.[33] Any clinical deterioration of the neurovascular status, as indicated by examination or ABIs, warrants an immediate surgical evaluation or diagnostic study (e.g., Doppler ultrasound or CT angiography) because this change may be secondary to an expanding popliteal thrombosis or compartment syndrome.

Anticoagulation

The use of anticoagulation and thrombolytics improves limb salvage and is recommended in patients with traumatic popliteal thrombosis.[34,35] It is contraindicated in patients with pelvic, intra-abdominal, or head trauma. The decision to begin systemic anticoagulation should be made in conjunction with orthopedic and vascular surgery consultants.

Prosthetic Knee Dislocations

Unlike prosthetic hip dislocations, prosthetic knee dislocations are very rare. Prosthetic knee dislocations can be posterior or anterior. (*See Figure 3.*)

Posterior dislocations typically occur in the post-operative period and are usually the result of trauma that disrupts the PCL ligament. Factors that predispose a person to posterior dislocations are valgus deformity of the knee, malposition or improper selection of prosthetic components, patellar instability, and extensor mechanism dysfunction. The mechanism for this dislocation is typically flexion and external rotation of the knee when the lateral side of the knee is too loose.[36]

Anterior dislocations more commonly occur months to years after surgery and usually are not associated with trauma.[36] Many of these dislocations result from loss of integrity of the posterior cruciate ligament, which provides anteroposterior stability of the knee and assists in femoral rollback. This motion is essential for the extensor mechanism of the knee to function. Stiehl et al postulated that abnormal kinetics of the prosthetic knee allow the PCL ligament to pull the femur posteriorly in the extended knee, resulting in anterior translation of the tibia and posterior translation of the femorotibial contact point. This abnormal motion causes gradual lengthening of the PCL and the posterior capsule, allowing the tibia to sublux and eventually dislocate anteriorly.[37]

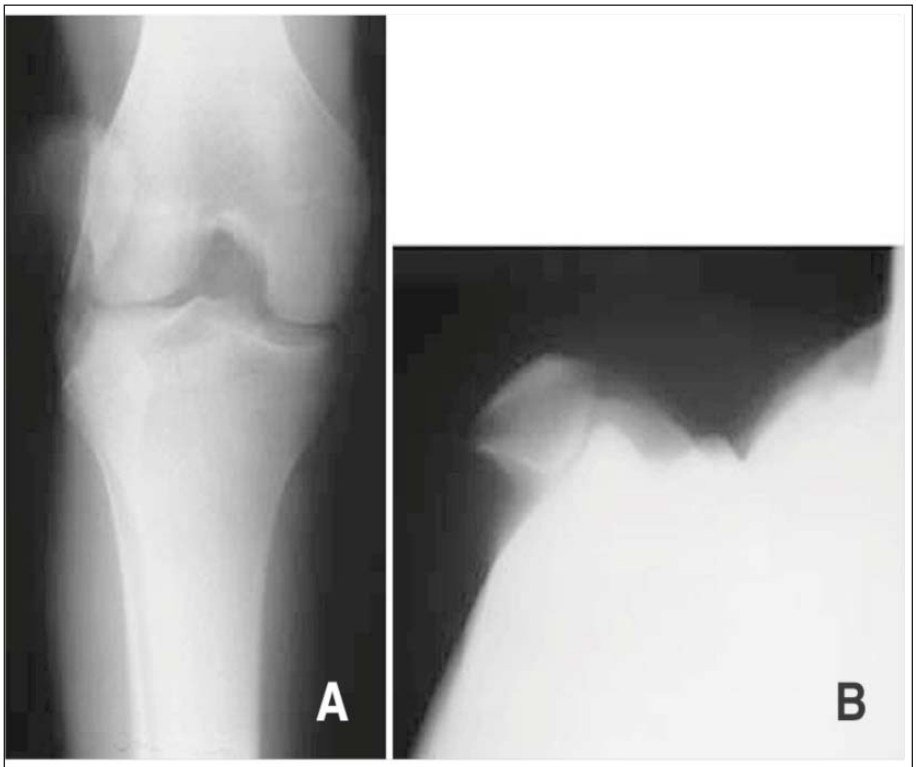
Prosthetic knee dislocations can be reduced in a similar manner to that used for native knee dislocations. These deformities should be evaluated in a similar manner to assess the patient for neurovascular injury. Some patients will require a revision of the knee prosthesis.

Patellar Dislocation

The patellofemoral joint consists of the articulation of the patella bone with the trochlear groove of the femur. The incidence of acute patellar dislocation in adults is estimated to be 20:100,000.[38] Females are at highest risk in the second decade of life, whereas males are at highest risk in the third decade of life.[17,38]

There are two types of patellar dislocation: lateral and medial. Lateral dislocations are more common. Dislocations can be caused by a direct blow or an indirect injury, with the latter being more common. Patellar dislocation from indirect injury can occur when the patient is standing with the knee flexed in a valgus position while the femur rotates internally on an externally rotated tibia, as can occur with the strong quadriceps muscle contraction of a baseball player's hind leg at the end of a swing.[38] This typically results in a lateral dislocation. A

Figure 4. Patellar Dislocation



A. Anteroposterior view; B. Sunrise view
Reproduced from: Minkowits R, Inzerillo C, Sherman O. Patella instability. *Bull NYU Hosp Jt Dis* 2007;65(4):280-293, with permission.

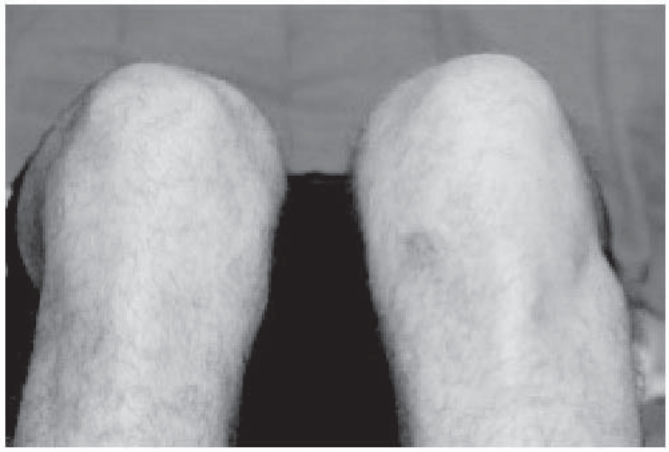
direct blow to the medial side of the patella can also force it laterally, causing a dislocation.

The medial patellofemoral ligament (MPFL) provides stability to the patellofemoral joint when the knee is between full extension (0 degrees) and 30 degrees of flexion.[17] Because it is the primary ligamentous structure preventing patella dislocation, it is also the ligamentous structure most commonly injured in acute patellar dislocations.[39] When the knee is flexed more than 30 degrees, the patella is protected from dislocation by the lateral femoral condyle and the trochlear groove.

Most acute patellar dislocations reduce spontaneously, which results in a diagnostic challenge for the clinician. The physical examination of the patella should begin with inspection. When the patient is sitting, the patella should be symmetric. Its normal position is centered within the trochlear groove and facing straight ahead. If the patella is in a high and lateral position, it is described as a "grasshopper eye," as it seems to look over the examiner's shoulder. (*See Figure 4.*) The patella is then palpated to localize areas of maximal tenderness, which suggests soft-tissue injury. Despite the diagnostic challenges with patellar dislocations, several physical examination findings are consistent with a patellofemoral dislocation: hemarthrosis (especially medially, owing to disruption of the MPFL and other medial restraints), tenderness over the medial facet of the patellar and adductor tubercle, medial ecchymosis, and a positive apprehension sign.

When acute patellar dislocation is suspected, a radiographic evaluation should be done to rule out osteochondral fragments within the joint. Anteroposterior (AP), lateral, and Merchant views should be obtained. The Merchant view is obtained by having the patient lie supine with the knee flexed to 45 degrees. The radiograph is taken with the X-ray beam aimed 30 degrees to the floor. A similar view is the sunrise view, which is obtained by having the patient lie prone with the knee acutely flexed.

Figure 5. Proximal Left Tibiofibular Joint Dislocation



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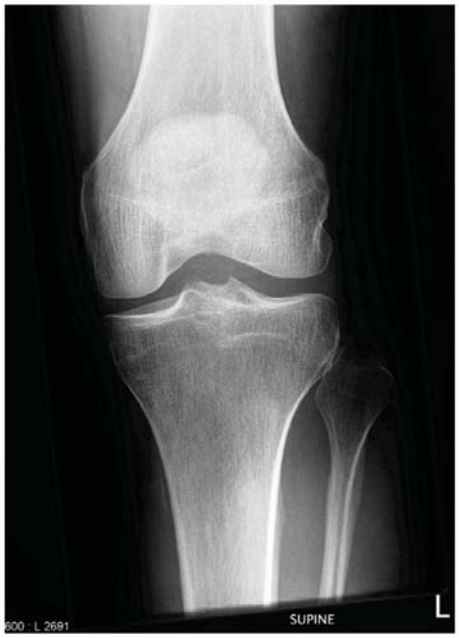
An MRI study can also be considered, as it is more sensitive for bony and cartilaginous pathology. MRI findings that are consistent with an acute patellofemoral dislocation are focal impaction injuries of the lateral femoral condyle, osteochondral injuries to the medial facet of the patella, and medial retinacular ligament injuries.[40] Associated osteochondral injuries are an indication for surgical repair; if this type of injury is missed, the patient's outcome is likely to be poor.

When the patella is in a "locked dislocation" (that is, it is still dislocated at this point in the evaluation), the clinician should attempt a closed reduction. A lateral patella dislocation can be reduced by extending the flexed knee while applying medially directed pressure to the lateral aspect of the patella. In many cases, the patella will be repositioned when the knee is fully extended or slightly hyperextended. The same technique can be used for medial dislocation, except the laterally directed pressure would be applied to the medial edge of the patella.

Indications for acute surgical management include persistent patellar subluxation and detachment of the vastus medialis obliquus muscle and medial retinaculum from the medial aspect of the patella. The ideal management (operative vs. non-operative) of primary, first-time patellar dislocations is a topic of debate. Nikku et al randomized 127 first-time lateral patellar dislocations without tibiofemoral ligamentous injuries or osteochondral fractures into two groups: closed treatment with immobilization and rehabilitation; and early operative treatment.[41] Outcome was assessed based on recurrent instability in the form of redislocation or recurrent subluxation, the need for additional operations, activity scores, and patients' opinions. Seven-year follow-up demonstrated no benefit of surgical intervention over immobilization and rehabilitation. However, surgically managed patients report significantly more sustained pain after operative repair.

Acute patellar dislocations can be managed conservatively with ice and elevation for the first several days and immobilization with a knee brace for 2 to 3 weeks. If a large hemarthrosis has formed, sterile aspiration is recommended prior to immobilization. Patients can bear weight as tolerated while the knee is in extension. Once the patient is comfortable, 6 weeks of rehabilitation is started.

Figure 6. Anteroposterior Radiograph of Left Knee Showing Lateral Displacement of Fibular Head



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Patients who experience recurrent subluxations or dislocations should be referred to an orthopedic surgeon for follow-up and consideration for surgical correction.

Proximal Tibiofibular Dislocation

The tibiofibular articulation is an inherently stable joint because it has tremendous ligamentous support, especially when the knee is in extension. Dislocation usually occurs when the knee is flexed while the foot is rotated and plantar-flexed. Isolated dislocations occur in sports that involve twisting of the knee.

Types of proximal tibiofibular dislocations include subluxation and anterolateral, posteromedial, and superior dislocations.[42] Subluxations tend to occur in pre-adolescent girls and are usually atraumatic. Anterolateral dislocations (*see Figures 5 and 6*)[43] are the most common type of this injury (85%) and usually result from a fall when the knee is flexed, the ankle is inverted, and the foot is plantar-flexed.[33]

Posteromedial dislocations (10%) are usually caused by direct trauma and are associated with common peroneal nerve injury.[33] Superior dislocations (2%) are uncommon and occur with high-energy ankle injuries. Superior dislocations are always associated with superior displacement of the lateral malleolus and are often associated with a common peroneal nerve injury.

Physical examination findings may be subtle. They include prominence of the fibular head, lateral knee pain, limited knee extension, and crepitus, popping, or locking. Normal radiographic characteristics of this joint include overlap of the fibular head with the lateral margin of the lateral tibial condyle on the AP view.[33] On the lateral view, most of the fibular head should lie posterior to the posterior margin of the tibia. CT is recommended as the study of choice for suspected proximal tibiofibular dislocations because it has higher sensitivities and specificities than plain radiographs.[42] Since findings on plain radiographs are subtle, a comparison radiograph of the contralateral limb may be indicated if the diagnosis is suspected and CT is unavailable.

Posteromedial dislocations are frequently associated with peroneal nerve injuries and are often irreducible because of interposed soft tissue. Anterolateral dislocations can be treated with closed reduction. This can be done by flexing the knee, dorsiflexing and externally rotating the foot, while placing pressure over the fibular head until a "pop" is heard or felt.[44] The knee should be immobilized initially, but the patient should be encouraged to begin early mobilization. Failed reduction of an anterolateral dislocation and of most posteromedial and superior dislocations requires open reduction by an orthopedist.

Conclusion

Knee and patellar dislocations have the potential to be devastating injuries with poor neurovascular outcomes. It is the emergency clinician's goal to identify patients who require emergent surgical intervention and to cautiously observe those in whom neurovascular injury might develop. With attention to detail and an understanding of the diagnostic and therapeutic options in the emergency setting, the emergency clinician can maximize the functional outcome of patients with these injuries.

Acknowledgement: The manuscript was copyedited by Linda J. Kesselring, MS, ELS, the technical editor/writer in the Department of Emergency Medicine at the University of Maryland School of Medicine.

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CME/CNE Questions

1. The most common type of knee dislocation is:
   A. anterior
   B. lateral
   C. medial
   D. posterior
   E. rotary
2. The risk of a popliteal artery injury in association with a knee dislocation is as high as:
   A. 10%
   B. 25%
   C. 40%
   D. 60%
   E. 80%
3. Popliteal artery injuries that are not treated within how many hours result in amputation in as many as 86% of patients, owing to ischemic changes?
   A. 12
   B. 14
   C. 16
   D. 8
   E. 10
4. What ankle brachial index is considered normal?
   A. greater than 0.0
   B. less than 0.5
   C. greater than 0.5
   D. less than 0.9
   E. greater than 0.9
5. After a knee dislocation has been reduced, it should be immobilized with the knee at what degree of flexion?
   A. 5 degrees
   B. 10 degrees
   C. 15 degrees
   D. 25 degrees
   E. 45 degrees

CNE/CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

6. The most common mechanism that causes a patellar dislocation is:
   A. a direct blow
   B. an indirect injury
7. The most common proximal tibiofibular dislocation is:
   A. an anterolateral dislocation
   B. a posteromedial dislocation
   C. a superior dislocation
8. How can lateral patella dislocations be reduced?
   A. By flexing the knee and applying lateral pressure to the medial border of the patella
   B. By flexing the knee and applying medial, pressure to the lateral border of the patella
   C. By hyperextending the knee and applying lateral to the medial border of the patella
   D. By hyperextending the knee and applying medial pressure to the lateral border of the patella
9. Which of the following is a hard sign of a vascular injury?
   A. capillary refill greater than 2 seconds
   B. diminished pulse
   C. history of hemorrhage
   D. pulsatile bleeding
   E. small or non-expanding hematoma
10. A Schenck Type IIIM knee dislocation is associated with a tear of which of the following ligaments?
   A. both cruciate ligaments and the lateral collateral ligament
   B. both cruciate ligaments and the medial collateral ligament
   C. both cruciate ligaments only
   D. one cruciate ligament and the lateral collateral ligament
   E. one cruciate ligament and the medial collateral ligament

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Vice President / Group Publisher: Donald R. Johnston

Executive Editor: Shelly Morrow Mark

Managing Editor: Leslie Hamlin

POSTMASTER: Send address changes to Trauma Reports, P.O. Box 105109, Atlanta, GA 30348.

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