Recognizing Eye Infections


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Every day, patients seek treatment for eye infections, which have a variety of causes and can affect any part of the eye. Eye infections usually are treated aggressively, because seemingly benign infections can quickly become serious and threaten vision. Symptoms that indicate an eye infection include chronic redness, persistent pruritus, flaking of the eyelid, eye discomfort or pain, blurred vision, eye discharge, and edema of ocular tissue. The following disease descriptions and case vignettes highlight a variety of eye infections, both common and uncommon, with which patients may present.

Conjunctivitis

Worldwide, conjunctivitis is an extremely common eye condition that has a variety of causes, including bacterial, viral, fungal, parasitic, allergic, toxic, and chemical. It is typically a self-limited process but can progress to a severe, sight-threatening infection, depending on the patient’s immune status and the cause.

Classic presentations generally involve eye discharge, pruritus, and a burning or gritty foreign-body sensation. Vision disturbances may be reported; however, this is more likely related to pus sliding across the eye than any actual declines in visual acuity.1 Bilateral disease suggests an infectious or allergic cause, whereas unilateral disease is more indicative of a toxic, chemical, mechanical, or lacrimal cause. Acute cases of conjunctivitis most often have a bacterial or viral cause.

Bacterial conjunctivitis is characterized by acute onset, minimal pain, and occasional pruritus. Thick and purulent discharge is common and generally copious. Chemosis is typical. Staphylococcal and streptococcal species are most often the causative pathogens, although Neisseria species, Chlamydia trachomatis, and other bacteria have been associated with this infection (Figures 1 and 2). Neisserial conjunctivitis is considered an ocular emergency and is indicative of systemic disease.

Viral conjunctivitis is characterized by acute or subacute onset, minimal pain, and exposure history. Pruritus and a clear, watery discharge are also common. Severe photophobia and foreign-body sensation sometime oc-
Adenovirus is usually the cause when the infection is associated with keratitis. If preauricular adenopathy and a follicular conjunctival change are present, the diagnosis is probably epidemic keratoconjunctivitis; however, herpes simplex virus and chlamydia can also cause these findings.

Diagnosis of conjunctivitis generally is based on history and physical examination. Laboratory tests are indicated in cases that do not improve within 48 to 72 hours after treatment has been initiated. Gram stain best determines the bacterial cause. If eosinophils are present, allergic conjunctivitis is probably the cause, although this finding can also indicate parasitic conjunctivitis. Culture and sensitivity testing should be performed for all neonates and immunosuppressed patients and when Neisseria gonorrhoeae is being considered as the cause.

Treatment is supportive. Antibiotic drops can help prevent a secondary bacterial infection, and topical antimicrobial therapy is recommended for all patients with suspected bacterial conjunctivitis. Topical sulfacetamide, erythromycin, gentamicin, ciprofloxacin, ofloxacin, and trimethoprim/sulfamethoxazole (TMP/SMX) are all reasonable choices. Neomycin-containing solutions should be avoided because of the risk of hypersensitivity reactions. Systemic treatment is required for gonococcal and chlamydial conjunctivitis.

**Toxoplasmic retinochoroiditis**

An 18-year-old woman presented with diminished vision and recent onset of floaters in the right eye. Mildly decreased visual acuity was noted in the right eye. There was no iritis, but dilated fundus evaluation indicated a vitritis—an area of retinal yellowish whitening with elevation and adjacent retinal vein inflammation (Figure 3). Toxoplasmic retinochoroiditis was diagnosed.

Unilateral and solitary ocular lesions that display little pigment deposition result from acute acquired toxoplasmosis, whereas congenital ocular toxoplasmosis is usually bilateral. Most ocular lesions are located in the posterior pole, particularly in the macular region. Ocular toxoplasmosis typically involves the inner retina and is associated with a marked vitreous reaction. Up to one-quarter of all posterior uveitis cases may be attributed to toxoplasmosis.

Toxoplasmosis is caused by *Toxoplasma gondii*. The common routes of transmission to humans are ingestion of contaminated food or undercooked meat, inhalation of the oocysts of the parasite shed in cat stool, and intrauterine infection of the fetus in an infected mother. This patient denied eating raw meat, but she did have several pet cats.

Acute lesions can heal spontaneously after several weeks or months. Treatment is indicated only if vision is adversely affected or the lesion is threatening the optic nerve or macula. Any of a number of antitoxoplasmic agents may be prescribed, including pyrimethamine, sulfadiazine, clindamycin, tetracycline, and TMP/SMX. Significant inflammation is treated with oral prednisone in conjunction with antimicrobial therapy. This patient’s infection responded to treatment with clindamycin, TMP/SMX, and prednisone.

**Periorbital streptococcal gangrene**

A 76-year-old woman with rheumatoid arthritis (RA) presented with a 1-day history of fever, chills, periorbital swelling, and eye discharge. During the course of a day, black discoloration of the skin developed around the orbits. She had a frontal headache but no sore throat or respiratory symptoms. Her treatment regimen for rheumatoid arthritis consisted of leflunomide and weekly oral methotrexate.

The patient’s blood pressure was 80/46 mm Hg and heart rate was 110 beats per minute. Her temperature was 37.7°C (99.8°F). She had periorbital swelling with skin necrosis, conjunctival hyperemia with purulent discharge, and normal fundi (Figure 4). On admission, her leukocyte count was 2300/µL, with 45% band forms and 33% segmented neutrophils. Cultures of blood and a peri-
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The orbital skin biopsy sample grew *Streptococcus pyogenes*. The patient was treated initially with broad-spectrum antibiotics and then on hospital day 2 with high doses of penicillin G and clindamycin. Her treatment regimen for RA was discontinued. She also underwent repeated surgical debridements. Unfortunately, the patient died as a result of nosocomial *Pseudomonas aeruginosa* septicemia on hospital day 36.

Invasive streptococcal infections of the skin and soft tissue have increased in frequency and severity over the past 20 years, but streptococcal gangrene involving the head and neck is rare. Prompt diagnosis, followed by antibiotic therapy and aggressive debridement, is paramount for a good outcome. The patient’s immunosuppressive therapy for RA probably predisposed her to this infection.

**Corneal ulcer**

A 55-year-old man who had undergone radial keratotomy and LASIK (laser-assisted in situ keratomileusis) surgery 6 years previously brushed his eye against a binocular eyepiece just before using his swimming pool and hot tub. He awoke the next morning with redness and pruritus of the eye with slight purulent discharge. Despite application of topical tobramycin, he noted later in the afternoon a mild foreign-body sensation in the eye and began treatment with topical gentamicin. The next morning, he noted worsened blurring and foreign-body sensation over 2 hours and onset of a severe right-sided headache.

On examination, vision was 20/240, compared with 20/20 previously, and the eye was markedly injected around the limbus, with slight corneal clouding. Slitlamp examination revealed a deep corneal ulcer at a healed radial keratotomy site, with corneal clouding and perilimbal injection as well as irregular progression of lamellar keratitis along the radial keratotomy scar (Figure 5).

Corneal scrapings grew *P. aeruginosa*. The patient was treated with topical ophthalmic solutions of vancomycin, tobramycin, ofloxacin, and ketorolac. At 1 week, the corneal ulcer was largely epithelialized and vision was 20/25. Topical ophthalmic ketorolac and ofloxacin solutions were continued for 1 more week, then tapered over 6 weeks, with complete resolution.

The patient’s previous radial keratotomy left him susceptible to infection even 6 years later, with only mild trauma and exposure to water as triggers.

**Ocular vaccinia**

Ocular vaccinia results from the transfer of vaccinia virus from a vaccination site or other lesion containing vaccinia virus to or near the eye (Figure 6). Ocular vaccinia accounts for most cases of inadvertent inoculation and usually manifests within 7 to 10 days of vaccination among first-time vaccinees. Persons at highest risk for inadvertent inoculation are children aged 1 to 4 years and persons with disruptions of the epidermis. In mild cases, the lesions are generally self-limited, resolving in about 3 weeks, and require no treatment. An ophthalmologist should be consulted in all suspected cases of ocular vaccinia because vision loss can occur.

Off-label use of the topical ophthalmic antiviral agents...
trifluridine and vidarabine can be considered for treatment of vaccinia involving the conjunctiva or cornea. To prevent spread to the conjunctiva and cornea if vaccinia lesions are present on the eyelid or lid margin, consider using these agents for prophylactic treatment. Topical antiviral therapy should be continued until all lesions have healed. Topical trifluridine is generally not used for longer than 14 days because it can cause superficial punctate keratopathy, which resolves after the medication is discontinued. Topical vidarabine may be preferable for use among children because it can be compounded into an ointment that allows for less frequent dosing and stings less than trifluridine does initially.

For severe ocular vaccinia, which involves marked hyperemia, edema, pustules, other focal lesions, lymphadenopathy, cellulitis, and fever, treatment with vaccinia immunoglobulin can speed recovery and prevent the spread of disease. If keratitis is present, topical ophthalmic antibacterials should be considered for prophylaxis of bacterial infection.

REFERENCES