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CME REVIEW ARTICLE

Diagnosis and management of pediatric conjunctivitis

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TARGET AUDIENCE

This CME activity is intended for physicians, nurse practitioners, and physician assistants who need to evaluate children for ophthalmologic complaints, diseases of the eye, and systemic illnesses with ocular manifestations and trauma. Specialists including pediatricians, emergency physicians, pediatric emergency physicians, family practitioners, allergists, and ophthalmologists will find this information especially useful.

OBJECTIVES

After completion of this article, the reader will be able to

1. Differentiate between allergic, bacterial, and viral conjunctivitis.
2. Identify the common pathogens in conjunctivitis in infants and children in various age brackets.
3. Discuss the treatment of bacterial conjunctivitis in newborns, infants, and older children.
4. Recognize the manifestations of herpetic keratitis and understand its treatment and complications.
5. Select the diagnosis and management of allergic conjunctivitis.

INTRODUCTION

One of the most common ophthalmologic complaints managed in the pediatric emergency department (ED) is conjunctivitis. Most commonly, conjunctivitis is due to bacterial infection, viral infec-

tion, or allergic hypersensitivity. The etiology of conjunctivitis varies with the age of the child (Table 1).

ETIOLOGY AND CLINICAL MANIFESTATIONS

Conjunctival inflammation may be a result of infection with bacterial or viral agents or allergic or toxic reactions. The signs and symptoms of conjunctivitis are similar with each of these etiologies (Table 2). The redness of conjunctivitis spares the limbus (1). If the limbus is involved, inflammation of the cornea (keratitis) or anterior segment (iritis or uveitis) should be considered (1). Ocular pain and photophobia are not typical of conjunctivitis (with the exception of adenoviral keratoconjunctivitis) and indicate a more serious disorder (2). Decreased visual acuity also suggests a more serious disease (1). Itching or the sensation of a foreign body are more likely due to allergic causes (1, 2). A clear or mucoid discharge may be associated with either allergic or viral etiologies (1–3), whereas a purulent discharge suggests a bacterial etiology (1–3).

Allergic conjunctivitis is usually seen in late childhood and early adulthood and is frequently associated with rhinitis, asthma, and eczema (2). Conjunctival edema and chemosis are also seen in allergic reactions (2). Preauricular adenopathy suggests a viral etiology, but this is not a specific finding (2, 3). An associated pharyngitis is frequently seen with adenovirus, especially in older children (1). Otitis media associated with conjunctivitis is most frequently caused by nontypeable *Haemophilus influenzae* and occasionally by *Streptococcus pneumoniae* and by other bacteria (4–6). Discrete lymphoid follicles encircled by blood vessels, a follicular response, can be seen with viral infections (3). A nonspecific or papillary response is more typical of bacterial infections (3). Papillae, in contrast to follicles, have a central vascular core (7). In one study, adenovirus was isolated from 20% of patients and from none of the control subjects; these children tended to be older, although there was considerable overlap in the age ranges (8).

BACTERIAL CONJUNCTIVITIS

Microbiology. The bacteria causing conjunctivitis vary with patient age. Neonates are more likely to have a bacterial etiology than older children. The most common microorganisms isolated from infants with conjunctivitis are *Chlamydia trachomatis*, *Staphylococcus*

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Key Words: Conjunctivitis, ophthalmia neonatorum, conjunctivitis-otitis syndrome, sexually transmitted diseases

TABLE 1
Diagnosis of conjunctivitis by age

Age groups	Common etiology	Treatment
Neonates	<24 hours	Chemical conjunctivitis
	<1 week	<i>Neisseria gonorrhoea</i>
	1–2 weeks	<i>Chlamydia trachomatis</i>
Infants and toddlers	Without otitis	<i>Haemophilus influenzae</i> , <i>Streptococcus pneumoniae</i> , <i>Branhamella catarrhalis</i>
	With otitis	<i>H. influenzae</i>
School-age children	1–5 years	HSV, varicella–zoster conjunctivitis
School-age children and adolescents		Viral conjunctivitis Allergic conjunctivitis
		Observation Hospitalize, ceftriaxone Oral erythromycin Topical antibiotics (Polysporin* or Polytrim†) Oral antibiotics Topical antivirals, oral acyclovir Supportive care, artificial tears Antihistamines, decongestants, H ₁ antagonists, mast cell stabilizers, NSAIDS

*Monarch Pharmaceuticals, Bristol, TN.

†Allergan, Irvine, CA.

HSV = herpes simplex virus; NSAIDS = nonsteroidal antiinflammatory drugs.

aureus, *Staphylococcus epidermidis*, *Viridans streptococci*, *Haemophilus* species, and *S. pneumoniae* (9–11). Since the institution of neonatal ocular prophylaxis, conjunctivitis due to *Neisseria gonorrhoea* is not often seen (9–12); however, it must remain in the differential diagnosis because of its potential to cause blindness and even death (1).

Bacterial etiologies are also more common in older children with nonepidemic conjunctivitis (3, 8, 13, 14). The most common pathogen in this age group is *H. influenzae*, followed by *S. pneumoniae* and *Moraxella catarrhalis* (3, 8, 13–15). *S. aureus* is frequently cultured from the eyes of normal children as well as from children with conjunctivitis (3, 13). Cultures positive for *S. aureus* may identify the etiologic agent, normal flora, or a contaminant from a concomitant blepharitis (3).

Ophthalmia Neonatorum. Conjunctivitis in the first month of life (ophthalmia neonatorum) is the most common infection in the neonatal period (9, 10, 16). In order of occurrence, the etiologies include chemical, chlamydial, bacterial, and viral (12). Conjunctivitis occurring within the first 24 hours of life is most likely an irritant reaction to ocular prophylaxis (16). This is most often seen with silver nitrate and occurs 2.5 to 12 times less often with erythromycin or tetracycline prophylaxis (16). The typical clinical course of chemical conjunctivitis is presentation of mild, purulent conjunctivitis within the first 24 hours of life and resolution within 48 hours (16). Gram stain of conjunctival scrapings shows no bacteria (16). No treatment is needed for this self-limited condition (16).

Many different species of bacteria have been reported to cause

ophthalmia neonatorum. The most important etiologic organism to recognize is *N. gonorrhoea*. An association between neonates with purulent conjunctivitis and mothers with vaginal discharge was first described in 1750 (11). Credé first described the use of topical silver nitrate to prevent ophthalmia neonatorum in 1884 (11, 16). Since the institution of neonatal ocular prophylaxis, the incidence of gonococcal conjunctivitis has decreased dramatically in the Western world (11, 12, 16). The typical presentation of gonococcal conjunctivitis is sudden, severe, grossly purulent conjunctivitis in the first 3 to 5 days of life (12, 16). If not recognized, this organism can rapidly progress within 24 hours to ulceration and perforation of the globe (16). If gonococcus is suspected, a culture and Gram stain of conjunctival scrapings should be obtained (12, 16). Concomitant cultures for chlamydia should also be obtained (16, 17). A presumptive diagnosis and treatment of gonococcal conjunctivitis is made based on the identification of gram-negative diplococci in conjunctival scrapings (16, 17). The most recently recommended treatment consists of hospitalization, to ensure adequate eye irrigation, and administration of ceftriaxone, 25 to 50 mg/kg intravenously or intramuscularly in a single dose, to a maximum of 125 mg (17, 18). Topical antibiotic therapy alone is not sufficient and is unnecessary (17, 18). The eyes should be irrigated with saline several times a day until the purulence subsides (16–18). The mother and her sexual contacts should be treated for gonorrhoea (16). The patient also should be evaluated for disseminated infection, such as arthritis, meningitis, or sepsis (17).

The most common identifiable organism causing ophthalmia

TABLE 2
Characteristics of bacterial, viral, and allergic conjunctivitis

	Bacterial	Viral	Allergic
Common etiologic agent	<i>Haemophilus influenzae</i> , <i>Streptococcus pneumoniae</i> , <i>Staphylococcus aureus</i> , <i>Neisseria gonorrhoea</i> , <i>Chlamydia trachomatis</i>	Adenovirus, HSV, HZV, enterovirus	Pollens, allergens
Prevalent age	Neonates to toddlers	School age to adults	Late childhood to early adulthood
Character of discharge	Purulent	Watery	Mucoid
Amount of discharge	1+ to 3+	1+ to 2+	1+
Injection	3+	2+	1+
Lymphadenopathy	Occasional	Common	None
Smear of exudate	Bacteria, PMNs	Lymphocytes	Eosinophils
Associated symptoms	Papillary response	Follicular response	Chemosis
Associated disorders	Otitis media	Pharyngitis, URI	Rhinitis, asthma, eczema
Treatment	Antibiotics	Artificial tears, acyclovir	Antihistamines, decongestants, mast cell stabilizers, NSAIDS

HSV = herpes simplex virus; HZV = herpes zoster virus; NSAIDS = nonsteroidal antiinflammatory drugs; PMNs = polymorphonuclear neutrophils; URI = upper respiratory infection.

neonatorum in the United States and Europe today is *C. trachomatis* (12, 16, 17). An infant born vaginally to a mother with an active chlamydial infection has approximately a 50% risk of acquiring the organism (12, 16, 19). Of these infants, approximately 25 to 50% will develop conjunctivitis (19). Chlamydial infections have also been described in infants born to mothers by cesarean section with intact membranes (12, 19). Unfortunately, neonatal ocular prophylaxis with silver nitrate, erythromycin, or tetracycline does not prevent the development of neonatal chlamydia conjunctivitis (12, 19, 20). The incubation period of chlamydia is typically 1 week, but it can vary from 5 to 14 days (16, 19). Thus, the clinical signs and symptoms of chlamydia conjunctivitis can develop a few days to several weeks after birth, most commonly at 2 weeks (12, 16, 19). However, any infant younger than 30 days with conjunctivitis should be considered to have a chlamydial etiology (17). The presentation can vary from mild to moderate conjunctival erythema and from scant, mucoid discharge to copious, purulent discharge; ocular edema, chemosis, or pseudomembrane formation may also be present (12, 16, 19). A diagnosis can be made by both culture and nonculture techniques (Table 3). Specimens for culture must be obtained using a Dacron-tipped swab, and epithelial cells must be collected, not just exudates (17, 19). Other tests using DNA probe, direct fluorescent antibody, enzyme immunoassay, polymerase chain reaction, and ligase chain reaction are also available and have high sensitivities and specificities (19). Chlamydia ophthalmia neonatorum is treated with oral erythromycin, 50 mg/kg/day divided into four daily doses for 10 to 14 days (17, 19). Sometimes a second course of erythromycin is needed, as the efficacy of erythromycin is approximately 80% (17, 19). If concomitant chlamydia pneumonia is present, it also can be treated with oral erythromycin (17, 19). One small study using azithromycin to treat chlamydia conjunctivitis looks promising, but further studies are needed before its use can be recommended (21). Topical antibiotics are ineffective and unnecessary (17, 19). The mother and her sexual contacts should be treated for chlamydia (17, 19). Untreated chlamydia conjunctivitis can result in varying degrees of conjunctival scarring and corneal infiltrates (16).

Many other organisms can cause ophthalmia neonatorum, including *S. aureus*, *S. pneumoniae*, viridans streptococci, other *Streptococcus* species, *H. influenzae*, *Escherichia coli*, *Pseudomonas* species, *Klebsiella* species, *Enterobacter* species, *Proteus* species, viruses, and others (9–12, 16). The role of *S. aureus* in neonatal conjunctivitis is controversial, as it is frequently cultured from the eyes of asymptomatic neonates (9–12, 16). Most infections caused by these organisms can be treated with topical antibiotics alone (16). *Pseudomonas aeruginosa* is the exception. Although infection with this organism is rare, *Pseudomonas* can cause corneal perforation, endophthalmitis,

blindness, and potentially death (16, 22). Pseudomonal conjunctivitis can present with edema, erythema of the eyelids, purulent discharge, pannus formation, or endophthalmitis, which can lead to sepsis, shock, or death (22). This diagnosis can be presumed when gram-negative rods are found in the exudate; growth of *Pseudomonas* in culture confirms the diagnosis (16). Because there is poor penetration of systemic and topical antibiotics into the anterior chamber, both systemic and topical aminoglycoside antibiotics are required (22, 23). Sometimes subconjunctival injections of the antibiotic are required (23, 24). All of these infants should be in strict isolation and should receive an ophthalmologic consultation (23, 24). Thus, once gonococcus, chlamydia, and *Pseudomonas* are ruled out, the recommendations for older children can be followed (12).

When the conjunctivae of neonates were cultured within 15 minutes of vaginal delivery in one study, the cultures grew coagulase-negative staphylococci, α -hemolytic streptococci, *Corynebacterium* species, *Propionibacterium* species, *Lactobacillus* species, *Bacteroides* species, and *Bifidobacterium* species (16). However, the cultures of infants born via cesarean section with membrane rupture occurring less than 3 hours earlier were sterile (16). If membrane rupture occurred more than 3 hours before cesarean section, the cultures grew intermediate bacterial populations (16).

Acute Bacterial Conjunctivitis. Beyond the neonatal period, acute conjunctivitis is twice as likely to be due to bacteria than to viruses (3, 8, 13, 14). *H. influenzae* is the most commonly isolated organism, followed by *S. pneumoniae* and *Branhamella catarrhalis* (formerly *Moraxella catarrhalis*) (3, 8, 13, 14). Staphylococcal species were isolated from the conjunctivae of children with conjunctivitis and from those of asymptomatic children at equal rates; thus, their role in the pathogenesis of conjunctivitis remains controversial (3, 8, 13, 14). Other bacteria were isolated from conjunctival cultures but at very small frequencies (13). The child may present with any of the following signs and symptoms: itching, burning, mucopurulent or purulent discharge, eyelid edema, or conjunctival erythema (3, 14). There are no pathognomonic signs to distinguish bacterial from viral conjunctivitis; however, there are some clues that may help differentiate the two (3, 14). Preschool-aged children are more likely to have bacterial etiologies, although there is considerable overlap in age ranges (14). The development of papillae, a papillary response, on the conjunctiva and bilateral disease are also more likely when the conjunctivitis is bacterial in origin (3, 14). Associated otitis media is highly suggestive of a bacterial etiology, which we will discuss more fully in the next section. However, given the significant overlap in the signs and symptoms of bacterial and viral conjunctivitis, clinicians cannot reliably predict etiology based on clinical examination (25). Gram stain of conjunctival exudates may be helpful, but some studies show poor sensitivity (25, 26).

TABLE 3
Methods for diagnosing chlamydia conjunctivitis

Method	Commercially available kit	Manufacturer
Culture		
Direct fluorescent antibody (DFA)	MicroTrak <i>Chlamydia trachomatis</i> Direct Specimen Test	Wampole Laboratories
Enzyme immunoassay (EIA)	Pathfinder Chlamydia Direct Specimen Test Intracel Bartels Chlamydia ELISA Kit MicroTrak II Premier Chlamydia Chlamydiazyme MicroTrak XL	Sanofi Pasteur Intracel Bartels Wampole Laboratories Meridian Diagnostics Abbott (semiautomated system) Wampole Laboratories (semiautomated system)
DNA probe	PACE 2	GenProbe
Nucleic acid amplification (ie, PCR, LCR)	Amplicor	Roche

LCR = ligase chain reaction; PCR = polymerase chain reaction.

A few uncommon pathogens need special mention because of their potential for harm. *N. gonorrhoea* is frequently considered in neonates but is often overlooked in the older child. As in neonates, this organism can cause ulceration and perforation of the cornea, leading to blindness (3). Patients present with abrupt onset of copious, purulent discharge; eyelid edema; and fever (3, 27, 28). Treatment consists of a single dose of ceftriaxone, 50 mg/kg intramuscularly (maximum of 1 g), and frequent irrigation of the eye with saline until purulence resolves (18). Because this organism is usually transmitted through sexual contact, all children with gonococcal conjunctivitis should be investigated for abuse. Alfonso et al. (28) described an outbreak of gonococcal conjunctivitis during an epidemic of acute hemorrhagic conjunctivitis caused by an enterovirus. These patients contracted the organism by using a folk remedy used to cure conjunctivitis: applying urine to the eyes (28). Sexually active adolescents certainly can contract gonococcal conjunctivitis; they also should be evaluated for other sites of infection as well as other concomitant sexually transmitted diseases (29).

Neisseria meningitidis is an uncommon ocular pathogen that can be associated with systemic meningococcal disease (3, 30). Meningococcal conjunctivitis was described as early as 1899 by Fraenkel (30, 31). Two types of meningococcal conjunctivitis have been described: primary, which can be invasive or noninvasive, and secondary, which occurs after systemic infection (30, 31). Complications of primary meningococcal conjunctivitis include corneal ulcers, keratitis, subconjunctival hemorrhage, iritis, and systemic meningococcal disease (30). Patients present with signs and symptoms similar to those of gonococcal conjunctivitis, with acute, gross, purulent discharge and sometimes fever (3, 30, 31). Approximately two thirds of cases are unilateral (3, 30). Gram stain of the conjunctival exudate shows gram-negative diplococci, and culture is diagnostic (3, 30–32). Systemic antibiotics should be administered; topical antibiotics are ineffective (30, 32).

Acute bacterial conjunctivitis is a self-limited disease, although it is frequently treated with topical antibiotics. In a double-blind, placebo-controlled study, children treated with either topical or systemic antibiotics experienced clinical improvement sooner and were able to eradicate bacteria from their conjunctivae, whereas those in the placebo group fared poorly (26). Therefore, although acute bacterial conjunctivitis is a self-limited disease, treatment with antibiotics is warranted for a bacterial cure and for a more rapid clinical cure. There are many topical antimicrobial agents available. Three inexpensive, commonly prescribed topical antibiotics, trimethoprim–polymyxin (Polytrim; Allergan, Irvine, CA), gentamicin, and sodium sulfacetamide, were compared in a double-blind study, which showed no difference in rate or speed of cure (33). In general, when choosing an antibiotic, one should consider the antibiotic's spectrum of activity, side effects, and cost. Bacitracin–polymyxin (Polysporin; Monarch Pharmaceuticals, Bristol, TN) and trimethoprim–polymyxin (Polytrim) are inexpensive, have few side effects, and have good broad-spectrum coverage (3, 34). Sodium sulfacetamide is inexpensive and has good gram-positive coverage, but it stings when applied (3, 34). There are case reports of Stevens-Johnson syndrome and systemic lupus erythematosus occurring after the use of ophthalmic sulfacetamide (35). Aminoglycosides have good gram-negative coverage, but they are expensive and cover streptococci poorly (3, 34). Epithelial toxicity and corneal ulceration can occur, especially with prolonged use of aminoglycosides (3, 34). Erythromycin is inexpensive and has good gram-positive and *Chlamydia* coverage, but it has poor activity against *Haemophilus* species, *B. catarrhalis*, staphylococcal species, and gram-negative organisms (3, 34). Fluoroquinolones are

expensive but have broad-spectrum coverage and few side effects other than local irritation (3, 34). Chloramphenicol is inexpensive and has broad-spectrum coverage, but there are case reports of associated aplastic anemia with topical ophthalmic administration (3, 35). Corticosteroids are sometimes combined with ophthalmic antibiotic preparations. Use of these agents should be avoided because the corticosteroid may impede eradication of the bacteria; worsen herpes keratitis, which may have been mistaken for conjunctivitis; and increase intraocular pressure (34). Thus, probably the best choice for initial empiric coverage is either bacitracin–polymyxin (Polysporin) or trimethoprim–polymyxin (Polytrim).

Clinicians are frequently asked about the need for isolation (ie, when can the child return to school). In a study comparing the strains of *H. influenzae* causing an outbreak of conjunctivitis in several day care centers, the strain of *H. influenzae* varied from child to child, even within the same day care center, suggesting low contagiousness (36). The strain of *H. influenzae* isolated from the conjunctiva matched the strain found in the child's nasopharynx, suggesting retrograde migration of the organism from the nasopharynx through the lacrimal duct to the conjunctiva (36). Thus, these children do not need to be isolated (37).

Conjunctivitis—Otitis Syndrome. The association between conjunctivitis and otitis media was first recognized by Coffey et al. (38) in 1966. Approximately one fourth of patients with conjunctivitis have concurrent otitis media, even in the absence of ear pain (6, 4, 9, 14, 26). Thus, all children with conjunctivitis need to be checked for otitis media. Cultures of the conjunctiva and middle ear fluid of children with conjunctivitis and otitis media have been found to grow predominately nontypeable *H. influenzae* and, less commonly, *S. pneumoniae* and other bacteria (4–6, 39). Bodor (4) also found a high incidence of purulent conjunctivitis and otitis media in siblings and playmates of index cases, suggesting a highly contagious nature of this infection. Because of the high frequency of β -lactamase in *H. influenzae*, therapy with antibiotics resistant to β -lactamase is recommended (39). Systemic antibiotics are needed to treat otitis media; thus, topical antibiotic therapy is not warranted (39).

VIRAL CONJUNCTIVITIS

Adenoviral Conjunctivitis. Most viral conjunctivitis is caused by adenovirus. Approximately 20% of all cases of conjunctivitis are caused by adenovirus, with a seasonal predilection for fall and winter months (9, 40). Several forms of adenoviral infection occur: follicular conjunctivitis, pharyngoconjunctival fever, epidemic keratoconjunctivitis, and, occasionally, acute hemorrhagic conjunctivitis (1, 3, 41). All forms of adenoviral conjunctivitis are extremely contagious. Transmission of infection is usually through direct contact with infected persons or contact with contaminated instruments (1, 3, 41). Thus, healthcare workers who manipulate the eyes should wear gloves and practice good hand-washing techniques (1, 3, 41, 42). Instruments used to examine patients should also be cleaned after use (1, 3, 41). Families must be instructed to separate the towels and bed sheets of the patient from other family members (1). The affected child should be kept home for approximately 1 week after the onset of symptoms (1, 41, 42). Treatment is supportive regardless of the type of adenoviral conjunctivitis (42, 43). Cold compresses, artificial tears, and topical vasoconstrictors may provide comfort (42–44). Studies comparing antiviral agents and antiinflammatory medications with artificial tears show no significant difference between these medications and artificial tears (44, 45). Topical steroids should be avoided because they have significant side effects, such as superinfection, glaucoma, and cataract (44, 46). Topical steroids also

may exacerbate a missed diagnosis of herpes conjunctivitis, may enhance adenoviral replication, and may increase the duration of adenoviral shedding (44, 46). Topical antibiotics are usually unnecessary, as secondary bacterial infections are rare (42).

Follicular Conjunctivitis. The most common type of adenoviral conjunctivitis produces a follicular reaction (41, 43). Follicles develop from an aggregation of lymphocytes and appear as small, pale, avascular areas surrounded by a network of blood vessels (3, 7, 41). Other findings on examination include preauricular lymphadenopathy, hyperemia, watery discharge, edema of the eyelids, rhinitis, pharyngitis, and other signs of upper respiratory tract infection (41, 43). The patient also may complain of itching and a foreign body sensation (43). A history of recent contact with another person with either a red eye or an upper respiratory tract infection is frequently present (43).

Pharyngoconjunctival Fever. Adenovirus types 3, 4, and 7 are most frequently implicated in pharyngoconjunctival fever (1, 3, 41, 43). Outbreaks have been linked to transmission of the virus in poorly chlorinated swimming pools and contaminated ponds (3, 41). Pharyngitis, fever, chemosis, hyperemia, and bilateral preauricular adenopathy are characteristic findings (1, 41, 43). Occasionally, small petechial hemorrhages in the conjunctiva are seen (41). It typically takes between 4 days and 2 weeks for symptoms to resolve (3, 41, 43).

Epidemic Keratoconjunctivitis. Adenovirus serotypes 8, 19, and 37 are most commonly responsible for epidemic keratoconjunctivitis (1, 3, 41, 43). Older children and adults are most commonly afflicted (3). Fomites contaminated with the virus can be infectious for up to 2 months (1). Although patients may have an associated upper respiratory tract infection, the ocular symptoms predominate. Symptoms include severe discomfort, photophobia, conjunctival edema, follicular (early) or papillary (late) response, and small petechial hemorrhages (3, 41, 43). Punctate epithelial defects also can be seen with fluorescein (43). Hazy, grayish-white subepithelial infiltrates can be found later in the course of illness (41, 43). These infiltrates may not resolve for several months (3, 41, 43). The conjunctival and eyelid swelling can be marked, and patients with severe cases may develop inflammatory pseudomembranes (3, 41, 43). Severe cases may be mistaken for preseptal or periorbital cellulitis (3, 43).

Acute Hemorrhagic Conjunctivitis. Acute hemorrhagic conjunctivitis is most commonly caused by picornaviruses, such as enterovirus and coxsackievirus, but can also be caused by adenovirus

type 11 (41, 43, 47). It is a highly contagious infection and usually occurs in epidemics (43). Most reported cases occur in Asia, but several epidemics have been reported in Africa, the Caribbean, South America, Central America, and Florida (43, 47). Patients present with sudden development of hyperemic conjunctiva, subconjunctival hemorrhages, chemotic conjunctiva, lid swelling, excessive tearing, photophobia, and pain (43, 47). The symptoms progress over 24 to 48 hours, last for 3 to 5 days, and then slowly resolve over 10 days (43, 47). Neurologic complications have been reported in cases due to enterovirus type 70 (43, 47, 48). The incidence is approximately one in 10,000, and the severity of neurologic symptoms ranges from transient, mild cranial nerve palsies to permanent flaccid paralysis (47, 48). Onset of neurologic symptoms occurs 10 to 20 days after development of conjunctivitis (47), and recovery time varies from less than a week to 10 weeks (48). Treatment is similar to that for adenovirus infections: supportive therapy with cold compresses, artificial tears, and topical decongestants (43, 47).

Herpes Virus Conjunctivitis. Herpes simplex virus (HSV) conjunctivitis may occur with primary infection or with recurrence. The peak age of primary infection is between 1 and 5 years (3). Recurrent infections typically occur in adults (43). Most ocular infections are due to HSV-1, except in neonates, in whom HSV-2 is more predominant (1). Transmission of the virus occurs through direct contact with another person who has an active lesion and is shedding virus or autoinoculation of the conjunctiva from lesions of a primary infection elsewhere (3). Characteristic findings include a follicular inflammatory response, serous discharge, and preauricular lymphadenopathy (1, 3). Other findings that may be present and help with the diagnosis include lid vesicles, upper respiratory tract infection, gingivostomatitis, and keratitis; also, approximately 80% of cases of HSV conjunctivitis are unilateral (1, 3, 43). Corneal involvement with the classic dendritic appearance is present in 50% of patients (1). Approximately 1.4 to 7% of cases of follicular conjunctivitis without corneal or lid signs are caused by HSV rather than adenovirus (49). Primary HSV infections are especially difficult to differentiate from adenoviral and chlamydial infections (42, 43, 49). The diagnosis can usually be made clinically, but if the diagnosis is in question, viral cultures and antigen detection tests can be performed (1, 3). However, the cells used to incubate HSV are different from those used to incubate adenovirus; therefore, the appropriate viral culture must be requested (49). Antigen detection tests are best performed from vesicle aspirates,

TABLE 4
Treatment of herpes simplex virus varicella-zoster virus conjunctivitis

Virus	Generic drug	Brand name drug	Route	Dosage
HSV	Trifluridine, 1%	Viroptic	Topical	1 drop in each eye every 2 hours until reepithelialized, then 1 drop every 4 hours for 7 days
	Idoxuridine, 0.1%	Herplex	Topical	1 drop in each eye every hour during day, every 2 hours during night; when improved, 1 drop every 2 hours during day and every 4 hours during night
	Vidarabine, 3%	Vira-A	Topical	Apply 0.5 inch to lower conjunctival sac 5 times per day every 3 hours while awake until reepithelialized, then twice daily for 7 days
VZV	Acyclovir	Zovirax	Intravenous	750 mg/m ² /day divided every 8 hours or 5 mg/kg/dose every 8 hours for 5–10 days
			Oral	80 mg/kg/day divided in 3–5 doses for 7–14 days
			Intravenous	5 mg/kg/dose every 8 hours for 5–10 days or 250 mg/m ² /dose every 8 hours for 5–10 days
			Oral	80 mg/kg/day divided 5 times per day for 5–7 days or 10–16 mg/kg/dose 5 times per day
			Oral	1000 mg three times a day for 7 days (≥12 years old)
	Famciclovir	Famvir	Oral	500 mg every 8 hours for 7 days (≥12 years old)
	Foscarnet	Foscavir	Intravenous	40 mg/kg every 8–12 hours for 14–21 days

HSV = herpes simplex virus; VZV = varicella-zoster virus.

TABLE 5
Treatment of allergic conjunctivitis

Type of agent	Generic drug	Brand name drug	Route	Dosage
Antihistamine/ decongestant	Naphazoline-antazoline	Vasocon-A	Topical	1-2 drops in each eye every 3-4 hours
	Naphazoline-pheniramine	Naphcon-A	Topical	1-2 drops in each eye every 3-4 hours
NSAID	Ketorolac, 0.5%	Acular	Topical	1 drop in each eye four times daily for up to 7 days
Mast cell stabilizer	Lodoxamide, 0.1%	Alomide	Topical	1-2 drops in each eye four times daily
	Nedocromil, 2%	Alocril	Topical	1-2 drops in each eye twice daily
	Pemirolast, 0.1%	Alamast	Topical	1-2 drops in each eye four times daily
	Cromolyn, 4%	Crolom	Topical	1 drop in each eye 4-6 times per day
	Emedastine, 0.05%	Emadine	Topical	1 drop in each eye four times daily
H ₁ receptor antagonist	Levocabastine, 0.05%	Livostin	Topical	1 drop in each eye four times daily
	Azelastine, 0.05%	Optivar	Topical	1 drop in each eye twice daily
Mast cell stabilizer/ H ₁ receptor antagonist	Ketotifen, 0.025%	Zaditor	Topical	1 drop in each eye every 8 to 12 hours
	Olopatadine, 0.1%	Patanol	Topical	1-2 drops in each eye twice daily

NSAID = nonsteroidal antiinflammatory drug.

but conjunctival scrapings can also be used at the cost of lower sensitivity (1, 49). An ophthalmologist should always be consulted in the treatment of HSV conjunctivitis (43, 50). Topical antiviral agents, such as trifluridine, iododeoxyuridine, or vidarabine, are typically used (Table 4) (1, 43, 50). Oral acyclovir can be used in severe cases or to suppress recurrent lesions (1, 50, 51). Steroids should be avoided, as they can aggravate the infection.

The conjunctiva can also be affected by varicella-zoster virus during primary or secondary infection (1, 43). Conjunctivitis is common in primary varicella (chickenpox), although corneal involvement is rare (1). Vesicles or ulcers are sometimes found on the bulbar or palpebral conjunctiva as well (1). Immunocompetent individuals do not typically need antiviral therapy for varicella (52). Secondary infection in the distribution of the ophthalmic division of the trigeminal nerve is known as *herpes zoster ophthalmicus*. The conjunctival findings are similar to those seen in HSV conjunctivitis, except there are vesicles in the trigeminal dermatome (1, 43). Viral cultures or immunofluorescence testing of the vesicle scrapings can be used in diagnosis (1, 52). However, the diagnosis is usually made based on clinical features (1). An ophthalmologist should be consulted in the treatment of herpes zoster ophthalmicus, as corneal involvement and iritis are common (1, 43). Oral or intravenous acyclovir is typically used to treat this condition in children (Table 4) (1, 52). Valacyclovir and famciclovir have been studied in adults with favorable results; however, their use in pediatric patients has not been studied (52, 53). Acyclovir-resistant varicella-zoster virus is treated with foscarnet (52). Treatment is best initiated within 72 hours of vesicle eruption (1, 53). Steroids are sometimes used to prevent permanent vision loss from glaucoma resulting from iritis; however, an ophthalmologist should always be consulted when determining a treatment regimen (1).

ALLERGIC CONJUNCTIVITIS

Allergic conjunctivitis is a chronic condition with frequent recurrences (54). Patients frequently have a history of other atopic disease, such as eczema, asthma, or, most commonly, rhinitis (2, 41, 42, 54). Peak age groups are late childhood and young adulthood (2). Symptoms may present seasonally or perennially (2, 42). An immunoglobulin E-mediated hypersensitivity reaction to an allergen produces the clinical manifestations (42). Symptoms include bilateral involvement, itching, tearing, mucoid discharge, redness, mild eyelid edema, and chemosis (2, 41, 42). Treatment consists of allergen avoidance, cold compresses, vasoconstrictors, antihistamines, topical nonsteroidal antiinflammatory agents, H₁-receptor antagonists, and mast cell stabilizers (Table 5) (41, 42, 55). Ophthalmic preparations of antihistamine-decongestant combinations are now available over the counter. These agents should only be used for a short time because continued use may cause a rebound hyperemia (2, 55). Topical ketorolac has been marketed to treat seasonal allergic conjunctivitis but can cause irritation on instillation and may not be as effective as the newer mast cell stabilizers (55, 56). Mast cell stabilizers, such as lodoxamide, nedocromil, and pemirolast, are effective in treating seasonal allergic conjunctivitis but may require a few days to a few weeks for improvement in symptoms (55, 57). H₁-receptor antagonists, such as emedastine and levocabastine, are also effective and have a more rapid onset of effect (55, 58). Ketotifen and olopatadine are both mast cell stabilizers and H₁-receptor antagonists and are also effective (55, 59). Few studies are available that compare these different classes of drugs; however, use of any of the drugs in the mast cell stabilizer, H₁-receptor antagonist, or the combination mast cell stabilizer and H₁-receptor antagonist groups should be effective in

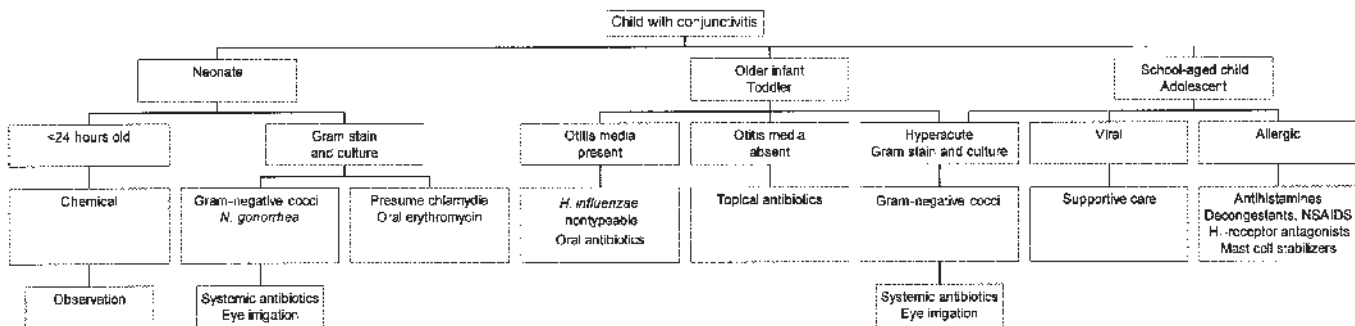


FIG. 1. Algorithm for evaluating conjunctivitis. NSAIDS = nonsteroidal antiinflammatory drugs.

providing symptom relief. The least expensive drugs in these groups are ketotifen and olopatadine (55).

CONCLUSIONS

The approach to a patient with conjunctivitis should begin by eliminating other causes of a red eye, such as iritis, keratitis, glaucoma, corneal abrasion, measles, Kawasaki disease, and others. The evaluation then varies with the age of the child (Fig. 1). If conjunctivitis develops in the first 24 hours of life, it is most likely due to chemical irritation from an agent used for prophylaxis of gonorrhea. However, all neonates with conjunctivitis should be evaluated for both *N. gonorrhoea* and *C. trachomatis* with Gram stains and culture. If gram-negative diplococci are seen, infection with gonorrhea is assumed and should be treated with systemic antibiotics. Infants younger than 30 days that do not have gonorrhea should be treated with oral erythromycin for presumed chlamydia infection. Mothers should be notified of positive culture results so that they themselves can be treated. Older infants and toddlers are more likely to have bacterial conjunctivitis. Their ears must be checked for otitis media, which, if present, should be treated with oral antibiotics. Patients with hyperacute conjunctivitis with rapidly progressive hyperemia, edema, and copious, purulent discharge should be evaluated for *N. gonorrhoea* and *N. meningitidis* with Gram staining and culture. Both of these organisms are treated with systemic antibiotics and frequent irrigation of the eyes. If the child does not have otitis media or hyperacute conjunctivitis, empiric topical antibiotics should be prescribed. Isolation is usually not needed for most cases of bacterial conjunctivitis, except those that involve *Neisseria* species and conjunctivitis-otitis media syndrome. Conjunctivitis in school-aged children and adolescents is most likely to be of viral or allergic origin. Associated findings that can help differentiate the two are preauricular lymphadenopathy, pharyngitis, upper respiratory tract infection, history of asthma, eczema or rhinitis, and history of recurrent conjunctivitis. Also, if any vesicles are seen in the vicinity, HSV or varicella-zoster virus is the likely etiologic agent. If either of these is suspected, a fluorescein examination of the eye must be performed to look for the characteristic dendritic pattern of herpes keratitis. Ophthalmologic consultation is needed for both herpes and zoster involvement of the conjunctiva. Other viral forms of conjunctivitis require only supportive care but are highly contagious and require approximately 1 week of isolation. Allergic conjunctivitis is not contagious, requires no isolation, and can be treated with antihistamines, decongestants, H₁-receptor antagonists, mast cell stabilizers, or nonsteroidal antiinflammatory drugs.

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