ELSEVIER
SAUNDERS

Infect Dis Clin N Am 21 (2007) 393–408

INFECTIOUS
DISEASE CLINICS
OF NORTH AMERICA

Periorbital and Orbital Infections

Ellen R. Wald, MD

Department of Pediatrics, University of Wisconsin School of Medicine and Public Health, Box 4108, 600 Highland Avenue, Madison, WI 53792, USA

Infections of the eye can be preseptal or orbital in origin (Box 1). They must be distinguished from noninfectious causes of swelling in or around the eye, including (1) blunt trauma (leading to the proverbial "black" eye), (2) tumor, (3) local edema, and (4) allergy. In cases of blunt trauma, history provides the key to the diagnosis. Eyelid swelling continues to increase for 48 hours and then resolves over several days. Tumors that characteristically involve the eye include hemangioma of the lid, ocular tumors such as retinoblastoma [1] and choroidal melanoma, and orbital neoplasms such as neuroblastoma and rhabdomyosarcoma [2]. Tumors usually cause gradual onset of proptosis in the absence of inflammation. Orbital pseudotumor, an autoimmune inflammation of the orbital tissues, presents with eyelid swelling, red eye, pain, and decreased ocular motility [3,4]. Hypoproteinemia and congestive heart failure cause eyelid swelling because of local edema. Characteristic findings are bilateral, boggy, nontender, nondiscolored soft tissue swelling. Allergic inflammation includes angioneurotic edema or contact hypersensitivity [5]. Superficially, these problems can resemble the findings in acute infection. The presence of pruritus and the absence of tenderness are helpful distinguishing characteristics of allergic inflammation.

Pathogenesis

Knowledge of the anatomy of the eye is important for understanding its susceptibility to spread of infection from contiguous structures. Veins that drain the orbit, the ethmoid and maxillary sinuses, and the skin of the eye and periorbital tissues (Fig. 1) constitute an anastomosing and valveless network [2]. This venous system provides opportunities for spread of infection from one anatomic site to another and predisposes to involvement of the

E-mail address: erwald@wisc.edu

0891-5520/07/$ - see front matter
doi:10.1016/j.idc.2007.03.008 *id.theclinics.com*

Box 1. Infectious causes of preseptal and orbital cellulitis

Preseptal cellulitis
Localized infection of the eyelid or adjacent structure
- Conjunctivitis
- Hordeolum
- Dacryoadenitis
- Dacryocystitis
- Bacterial cellulitis (trauma)

Hematogenous dissemination
- Bacteremic periorbital cellulitis

Acute sinusitis
- Inflammatory edema

Orbital cellulitis
Acute sinusitis
- Subperiosteal abscess
- Orbital abscess
- Orbital cellulitis
- Cavernous sinus thrombosis

Hematogenous dissemination
- Endophthalmitis

Traumatic inoculation
- Endophthalmitis

cavernous sinus. Bacterial cellulitis of the soft tissue of the facial structures can cause contiguous phlebitis that may progress to involve distant sites.

Fig. 2 demonstrates the relationship between the eye and the paranasal sinuses. The roof of the orbit is the floor of the frontal sinus, and the floor of the orbit is the roof of the maxillary sinus. The medial wall of the orbit is formed by the frontal maxillary process, the lacrimal bone, the lamina papyracea of the ethmoid bone, and a small part of the sphenoid bone [6]. Infection originating in the mucosa of the paranasal sinuses can spread to involve the bone (osteitis with or without subperiosteal abscess) and the intraorbital contents. Orbital infection can occur through natural bony dehiscences in the lamina papyracea of the ethmoid or frontal bones or through foramina through which the ethmoidal arteries pass [5].

Fig. 3 shows the position of the orbital septum. This structure is a connective-tissue extension of the periosteum (or periorbita) that is reflected into the upper and lower eyelids. Infection of tissues anterior to the orbital septum is described as ''periorbital'' or ''preseptal'' [7]. The septum provides a nearly impervious barrier to spread of infection to the orbit. Although preseptal cellulitis or periorbital cellulitis (the terms can be used

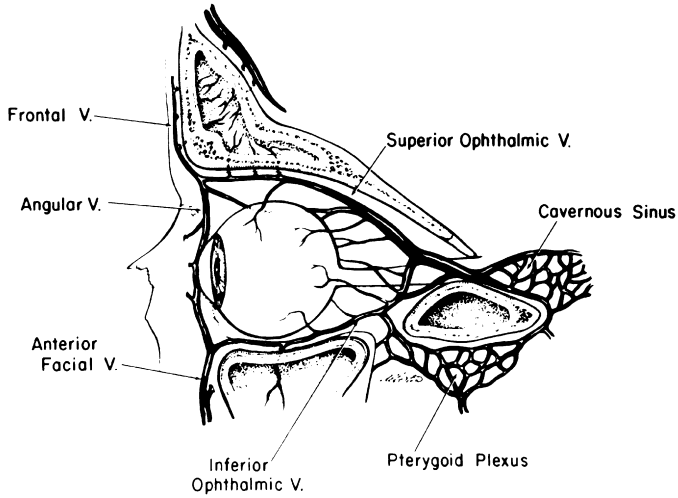

Fig. 1. The valveless venous system of the orbit and its many anastomoses. V, vein. (*From* Harris GJ. Subperiosteal abscess of the orbit. Arch Ophthalmol 1983;101:753–4; with permission.)

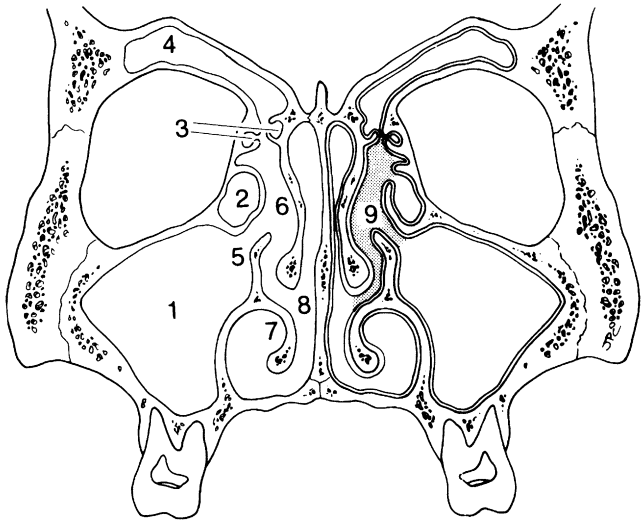

Fig. 2. The relationship between the eye and the paranasal sinuses is shown schematically. The roof of the orbit, the medial wall, and the floor are shared by the frontal, ethmoid, and maxillary sinuses, respectively. 1, maxillary sinus; 2, ethmoidal bulla; 3, ethmoidal cells; 4, frontal sinus; 5, uncinate process; 6, middle turbinate; 7, inferior turbinate; 8, nasal septum; 9, osteomeatal complex. (*From* Shapiro ED, Wald ER, Brozanski BA. Periorbital cellulitis and paranasal sinusitis: a reappraisal. Pediatr Infect Dis J 1982;1:91–4; with permission.)

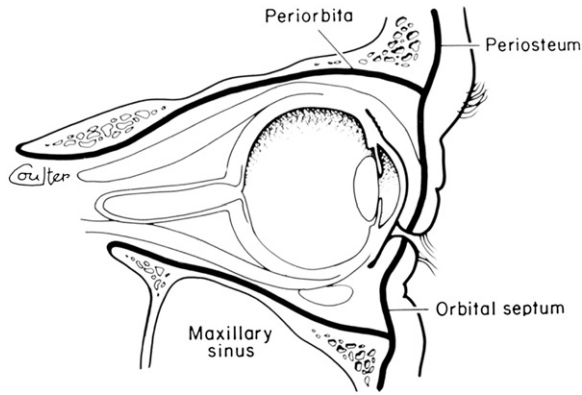

Fig. 3. The orbital septum is a connective tissue extension of the periosteum that is reflected into the upper and lower lid. (*From* Shapiro ED, Wald ER, Brozanski BA. Periorbital cellulitis and paranasal sinusitis: a reappraisal. Pediatr Infect Dis J 1982;1:91–4; with permission.)

interchangeably) often is considered a ''diagnosis,'' the term is an inadequate diagnostic label unless accompanied by a modifier that indicates likely pathogenesis.

Infectious causes of preseptal cellulitis occur in the following three settings: (1) secondary to a localized infection or inflammation of the conjunctiva, eyelids, or adjacent structures (eg, conjunctivitis, hordeolum, acute chalazion, dacryocystitis, dacryoadenitis, impetigo, traumatic bacterial cellulitis); (2) secondary to hematogenous dissemination of nasopharyngeal pathogens to the periorbital tissue; and (3) as a manifestation of inflammatory edema in patients who have acute sinusitis (see Box 1) [7].

Infections behind the septum that cause eye swelling include subperiosteal abscess, orbital abscess, orbital cellulitis, cavernous sinus thrombosis, panophthalmitis, and endophthalmitis. Although all these entities can be labeled ''orbital cellulitis,'' a systematic approach allows a more specific diagnosis, thereby directing management.

Preseptal infections

Conjunctivitis

Conjunctivitis is the most common disorder of the eye for which children are brought for medical care. In most cases, the lids are crusted and thickened with hyperemic conjunctiva. The usual causes of conjunctivitis in children older than neonates but less than 6 years old are *Haemophilus influenzae* (nontypeable) and *Streptococcus pneumoniae* [8–10]. Acute otitis media is a complicating feature in approximately 20% to 25% of children who have conjunctivitis caused by *H influenzae* [11]. In this case, systemic

antibiotics are preferable to topical ophthalmic preparations for treatment. Several large outbreaks of conjunctivitis caused by an unencapsulated strain of *S pneumoniae* have been reported recently among college students [12]. These organisms also may cause sporadic cases of conjunctivitis [13]. In cases of bacterial conjunctivitis without acute otitis media, topical therapy with polymyxin-bacitracin [14], ciprofloxacin [15], norfloxacin [16], and chloramphenicol [17] hastens resolution [18].

Adenovirus is the most common cause of viral conjunctivitis in children older than 6 years [19]. Occasionally, individuals who have adenovirus infection have diffuse swelling of the lids that can be mistaken for a more serious problem (Fig. 4) [20]. To establish the microbiologic diagnosis in patients who have conjunctivitis, a swab of the conjunctival surface should be obtained for bacterial and viral pathogens.

Hordeolum and chalazion

An external hordeolum, or stye, is a bacterial infection of the glands of Zeis or Moll (sebaceous gland or sweat gland, respectively) associated with a hair follicle on the eyelid. In most cases, infection is localized and points to the lid margin as a pustule or inflammatory papule. The lid can be slightly swollen and erythematous around the area of involvement. An external hordeolum usually lasts a few days to a week and resolves spontaneously.

An internal hordeolum is a bacterial infection of a meibomian gland, a long sebaceous gland whose orifice is at the lid margin [21]. The infection usually causes inflammation and edema of the neck of the gland, which can result in obstruction. If there is no obstruction, infection points to the lid margin. If obstruction is present, infection points to the conjunctival surface of the eye [21]. Sometimes the swelling caused by an acute internal hordeolum is diffuse rather than localized, and a pustule is not obvious on the lid margin. To clarify the cause, it is necessary to evert the eyelid and examine the tarsal conjunctiva. A tiny, delicate pustule is diagnostic of an internal hordeolum.

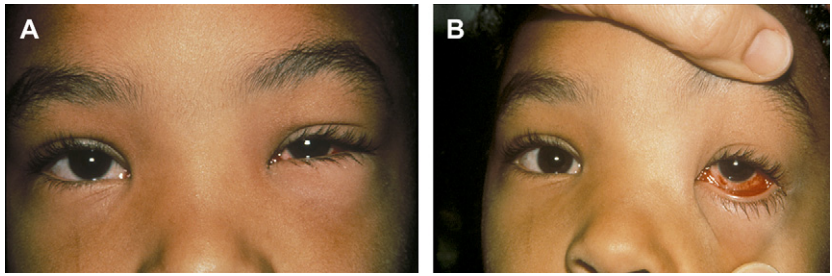

Fig. 4. (*A* and *B*) A child who has hemorrhagic conjunctivitis caused by adenovirus.

The usual cause of acute internal or external hordeola is *Staphylococcus aureus* [22]. An antibiotic ophthalmic ointment containing bacitracin can be applied to the site of infection. The main purpose of the topical therapy is to prevent spread of infection to adjacent hair follicles. Warm compresses may facilitate spontaneous drainage.

In contrast to the internal hordeolum, a chalazion manifests as a persistent (more than 2 weeks in duration), nontender, localized bulge or nodule (3–10 mm) in the lid; the overlying skin is completely normal. It is a sterile lipogranulomatous reaction. When a chalazion is large and causes local irritation, incision may be required.

Dacryoadenitis

Dacryoadenitis is an infection of the lacrimal gland. Sudden onset of soft tissue swelling that is maximal over the outer portion of the upper lid margin is typical. Occasionally, the eyeball is erythematous, the eyelid swollen, and the patient can have remarkable constitutional symptoms. The location of the swelling is a distinguishing characteristic (Fig. 5). When dacryoadenitis is caused by viral infection (mumps virus, Epstein-Barr virus [23], cytomegalovirus, coxsackievirus, echoviruses, or varicella-zoster virus), the area is only modestly tender. By contrast, when the infection is caused by bacterial agents, discomfort is prominent. In addition to *S aureus,* which is the most common cause of bacterial dacryoadenitis, causative agents include streptococci, *Chlamydia trachomatis, Brucella melitensis,* and, occasionally, *Neisseria gonorrhoeae* [24]. Fungal and rare parasitic infections of the lacrimal gland have been reported, including *Cysticercus cellulosae* and *Schistosoma haematobium* [25].

If parenteral therapy is required for suspected bacterial dacryoadenitis caused by *S aureus*, nafcillin, 150 mg/kg/d divided into doses every 6 hours,

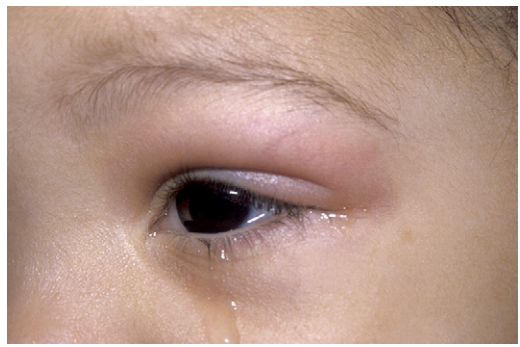

Fig. 5. A patient who has dacryoadenitis caused by an unspecified viral infection. The nontender swelling over the lateral portion of the left upper lid evolved while an antibiotic was being administered for acute otitis media. Swelling resolved in several days without any change in medical treatment.

is appropriate. In circumstances in which methicillin-resistant *S aureus* (MRSA) is a concern, vancomycin, 40 mg/kg in three to four divided doses, is recommended. Oral treatment of acute dacryoadenitis is undertaken with a semisynthetic penicillin such as dicloxacillin (100 mg/kg/d divided into four doses), cephalexin, or cefadroxil (100 or 50 mg/kg/d, respectively, divided into doses every 6 or 12 hours, respectively). If MRSA is suspected, alternatives include sulfamethoxazole-trimethoprim (based on 40 mg/kg/d of trimethoprim in two doses), clindamycin (40 mg/kg/d divided into doses every 6 hours), or linezolid (20 mg/kg/d divided into doses every 12 hours). Treatment is continued until all signs and symptoms have disappeared.

The differential diagnosis of swelling of the upper outer aspect of the eyelid includes inflammatory noninfectious problems such as Sjögren's syndrome and sarcoidosis as well as benign and malignant tumors [25].

Dacryocystitis

Dacryocystitis is a bacterial infection of the lacrimal sac. Although it is uncommon, it can occur at any age as a bacterial complication of a viral upper respiratory tract infection (URI). Because of the course traversed by the lacrimal duct, which drains to the inferior meatus within the nose, it is surprising that the duct and sac are not infected more often. Delayed opening, inspissated secretions, and anatomic abnormalities lead to disproportionate representation of infants younger than 3 months among children who have dacryocystitis [26].

Patients who have dacryocystitis often have had a viral URI for several days. They then experience fever and impressive erythema and swelling in addition to exquisite tenderness, which is most prominent in the triangular area just below the medial canthus (Fig. 6). Pressure over the lacrimal sac causes considerable discomfort but can result in expression of purulent material from the lacrimal puncta. Common causative organisms are gram-positive cocci. *S pneumoniae* is most common in neonates, although *S aureus, H influenzae,* and *Streptococcus agalactiae* also have been reported

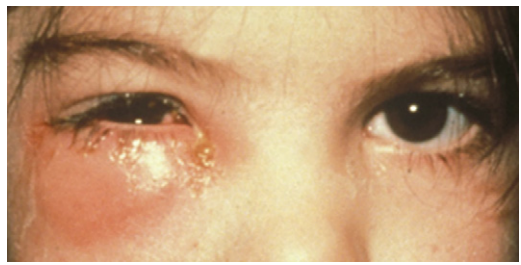

Fig. 6. A 6-year-old girl who has dacryocystitis. The area beneath the medial canthus is erythematous, indurated, and exquisitely tender.

[26,27]. *S aureus* and *Staphylococcus epidermidis* are most commonly implicated in acquired dacryocystitis in the older patient [28]. It is important to obtain material from the punctum, because other organisms (including enteric gram-negative bacilli, anaerobic bacteria, and yeast) have been observed occasionally [29]. Unusual pathogens, such as *Pasteurella multocida* and *Aeromonas hydrophila,* have been reported rarely [30].

Most patients who have dacryocystitis require admission to the hospital. Often they appear ill or toxic [29]. Because of the potential for any case of bacterial facial cellulitis to result in cavernous sinus thrombosis, therapy with parenteral antibiotics is indicated until the infection begins to subside. Nafcillin (at a dose of 150 mg/kg/d divided into doses every 6 hours) or cefazolin (at a dose of 100 mg/kg/d divided into doses every 8 hours) is appropriate except when infection caused by MRSA is suspected. In the latter case, vancomycin (40 mg/kg/d divided into doses every 6 hours) is best initiated. In penicillin-allergic patients, vancomycin or clindamycin (40 mg/kg/d divided into doses every 6 hours) suffices. After substantial improvement is observed in local findings, an oral agent can be substituted to complete a 10- to 14-day course of therapy.

The role of nonmedical management of dacryocystitis is controversial. Although surgical manipulation of the lacrimal duct is not necessary for most patients, both probing of the duct and incision and drainage have been reported to be successful in neonates [27]. Incision and drainage and direct application of antibiotics inside the sac have been promoted by some practitioners who care for adults [31].

Preseptal cellulitis after trauma

Occasionally, preseptal cellulitis results from secondary bacterial infection of sites of local skin trauma (including insect bites) or with spread of infection from a focus of impetigo. The traumatic injury may be extremely modest or completely unapparent. Loosely bound periorbital soft tissues permit impressive swelling to accompany minor infection. The overlying skin can be bright red with subtle textural changes, or intense swelling can lead to shininess (Fig. 7). Some patients have fever, but many are afebrile despite dramatic local findings. The peripheral white blood cell count is variable. In these cases, cellulitis, similar to that on any other cutaneous area, is caused by *S aureus* (including MRSA) or group A streptococcus [32,33].

Several less common causes of lid cellulitis have been reported. Periocular cellulitis and abscess formation have resulted from infection with *P multocida* in a healthy child who sustained a cat bite and cat scratch to the eyelid [34]. Ringworm (caused by *Trichophyton* species) also has been recognized as a cause of lid infection (leading to preseptal cellulitis) characterized by redness, swelling, ulceration, and vesicle formation [35,36]. Palpebral myiasis involving the eyelid of a 6-year-old child was reported from the Massachusetts Eye and Ear Infirmary [37]. A small draining fistula through which

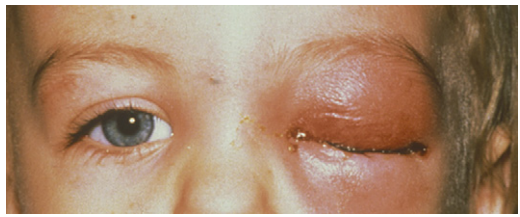

Fig. 7. A 3-year-old boy had rapid onset of left-eyelid swelling and erythema after he incurred a small laceration at the lateral margin of the left eye. He had had an upper respiratory tract infection for 10 days. Group A streptococcus was recovered from the wound.

the larvae were extracted was noted at the site of the erythematous and edematous lid. Several cases of cellulitis of the eyelid caused by *Bacillus anthracis* have been reported from Turkey [38]. The diagnosis was suspected when the erythematous and swollen lid developed an eschar. Scrapings showed the presence of gram-positive rods that were confirmed by culture. A primary case of lymphocutaneous *Nocardia brasiliensis* of the eyelid has been reported in an adult who was hunting in England 2 weeks before presentation following a small abrasion on this lower eyelid [39]. In countries where *Mycobacterium tuberculosis* is endemic, this cause also should be considered in patients who present with a swollen lid. Raina and colleagues [40] reported seven children who had tuberculous lesions of their eyelids. In most cases the presentation was relatively indolent (2 days to 2 months), and fistulas occurred during the course of conventional antibiotic treatment for more typical bacterial disease. Diagnosis was confirmed by a positive tuberculin skin test, the identification of a primary focus of tuberculosis in lung or bone, and response to antituberculous therapy.

Patients who have bacterial cellulitis of traumatized areas rarely have bacteremia. Precise bacteriologic diagnosis is made through culture of exudate from the wound. If there is no drainage, a careful attempt at tissue aspiration is undertaken if it can be done safely (ie, far enough from the orbit that there can be no potential damage to the eye). A tuberculin syringe with a 25-gauge needle can be used for aspiration of "tissue juice." Usually, only a minuscule amount of infected material can be aspirated. A small volume of nonbacteriostatic saline (0.2 mL) is drawn into the syringe before the procedure. The saline is not injected into the skin; instead, it is used to expel the small volume of tissue fluid onto chocolate agar for culture [41]. In patients who have bacterial cellulitis, parenteral treatment similar to that advised for dacryocystitis is recommended to hasten resolution and avoid spread of infection to the cavernous sinus.

Bacteremic periorbital cellulitis

Bacteremic periorbital cellulitis, most often seen in infants younger than 18 months of age, is preceded by a viral URI for several days. There is a sudden increase in temperature (to $>39^{\circ}C$) accompanied by the acute onset and

rapid progression of eyelid swelling. Swelling usually begins in the inner canthus of the upper and lower eyelid and can obscure the eyeball within 12 hours. Periorbital tissues are markedly discolored and usually are erythematous, although the area may have a violaceous discoloration if the swelling has been rapidly progressive [42,43]. The child's resistance to examination commonly leads to the erroneous impression of tenderness. Retraction or separation of the lids reveals that the globe is normally placed and extraocular eye movements are intact. If retraction of the lids is not possible, orbital CT scan may be necessary [44]. The young age, high fever, and rapid progression of findings differentiate bacteremic preseptal cellulitis from other causes of swelling around the eye.

In the era before universal *H influenzae* type b (Hib) immunization, this organism was the most common cause of bacteremic periorbital cellulitis in approximately 80% of cases [45]; *S pneumoniae* accounted for the remaining 20%. The substantial decline that has been observed in the total number of cases of bacteremic periorbital cellulitis is attributable to the widespread use of the Hib vaccine since 1991 and the introduction of pneumococcal conjugate vaccine in 2000 [45,46]. A precise bacteriologic diagnosis is made by recovery of the organism from blood culture. If a careful tissue aspiration is performed, culture of the specimen may have a positive result.

The pathogenesis of most of these infections, which usually occur during the course of a viral URI, is hematogenous dissemination from a portal of entry in the nasopharynx. This process is akin to the mechanism of most infections caused by Hib and some infections caused by *S pneumoniae*. In the current era, with routine immunization for *H influenzae* and *S pneumoniae,* these infections are rare [47].

In patients who have bacteremic periorbital cellulitis, radiographs of the paranasal sinuses often are abnormal. The abnormalities, however, almost certainly reflect the viral respiratory syndrome that precedes and probably predisposes to the bacteremic event, rather than a clinically significant sinusitis [7]. Bacteremic cellulitis rarely arises from the paranasal sinus cavities, as evidenced by the finding that typeable *H influenzae* organisms almost never are recovered from maxillary sinus aspirates and likewise rarely are recovered from abscess material in patients who have serious local complications of paranasal sinus disease, such as subperiosteal abscess. Although *S pneumoniae* can cause subperiosteal abscess in patients who have acute sinusitis, such patients usually are not bacteremic.

Treatment for suspected bacteremic periorbital cellulitis requires parenteral therapy. A nonvaccine strain of *S pneumoniae* or *Streptococcus pyogenes* is the most likely cause in a child who has received both the Hib and heptavalent pneumococcal conjugate vaccine [48]. Because this infection is usually bacteremic in the age group in whom the meninges are susceptible to inoculation, it may be prudent to use an advanced-generation cephalosporin such as cefotaxime or ceftriaxone (150 or 100 mg/kg/d, respectively, divided into 8- or 12-hour doses, respectively). Lumbar puncture should be performed unless

the clinical picture precludes meningitis. Addition of vancomycin (60 mg/kg/d divided into doses every 6 hours) or rifampin (20 mg/kg once daily, not to exceed 600 mg/d) is appropriate if cerebrospinal fluid pleocytosis is present. When evidence of local infection has resolved and there is no meningitis, oral antimicrobial therapy is prescribed to complete a 10-day course.

Preseptal (periorbital) cellulitis caused by inflammatory edema of sinusitis

Several complications of paranasal sinusitis can result in the development of swelling around the eye. The most common and least serious complication often is referred to as ''inflammatory edema'' or a ''sympathetic effusion'' [6]. This complication is a form of preseptal cellulitis, although infection is confined to the sinuses [49].

Typically, a child at least 2 years old has had a viral URI for several days when swelling is noted. Often, there is a history of intermittent early morning periorbital swelling that resolves after a few hours. On the day of presentation, the eyelid swelling does not resolve typically but progresses gradually. Surprisingly, striking degrees of erythema can also be present. Eye pain and tenderness are variable. Eyelids can be very swollen and difficult to evert, requiring the assistance of an ophthalmologist. There is no displacement of the globe or impairment of extraocular eye movements, however. Fever, if present, is usually low grade.

The peripheral white blood cell count is unremarkable. Blood culture results are always negative. If a tissue aspiration is performed, culture of the specimen has a negative result. Sinus radiographs show ipsilateral ethmoiditis or pansinusitis. The age of the child, gradual evolution of lid swelling, and modest temperature elevation differentiate inflammatory edema from bacteremic periorbital cellulitis.

The pathogenesis of sympathetic effusion or inflammatory edema is attributable to the venous drainage of the eyelid and surrounding structures. The inferior and superior ophthalmic veins, which drain the lower lid and upper lid, respectively, pass through or just next to the ethmoid sinus. When the ethmoid sinuses are completely congested, physical impedance of venous drainage occurs, resulting in soft tissue swelling of the eyelids, maximal at the medial aspect of the lids. In this instance, infection is confined within the paranasal sinuses. The globe is not displaced, and there is no impairment of the extraocular muscle movements. Inflammatory edema, however, is part of a continuum with more serious complications resulting from the spread of infection outside the paranasal sinuses into the orbit [50]. Rarely, infection progresses despite initial optimal management of sympathetic effusions [49].

The infecting organisms in cases of inflammatory edema are the same as those that cause uncomplicated acute sinusitis (ie, *S pneumoniae,* nontypeable *H influenzae,* and *Moraxella catarrhalis*). Antibiotic therapy can be given orally if, at the time of the first examination, the eyelid swelling is

modest, the child does not appear toxic, and the parents will adhere to management. Otherwise, admission to the hospital and parenteral treatment should be undertaken.

The only potential source of bacteriologic information is that which may be obtained by maxillary sinus aspiration; however, this procedure is rarely performed if infection is confined to the sinuses. Outpatient treatment should be considered only if the eyelid is at least 50% open and very close follow-up can be assured. Appropriate agents for outpatient therapy have activity against beta-lactamase–producing organisms (eg, amoxicillin–potassium clavulanate, cefuroxime axetil, and cefpodoxime proxetil). Parenteral agents include cefuroxime (150 mg/kg/d divided into doses every 8 hours) and ampicillin-sulbactam (200 mg/kg/d divided into doses every 6 hours). The latter combination, although not approved for children younger than 12 years, is an attractive choice. Although the use of topically applied intranasal decongestants such as oxymetazoline has not been evaluated systematically, such agents may be helpful during the first 48 hours. After several days, once the affected eye has returned to near normal, an oral antimicrobial agent is substituted to complete a 14-day course of therapy.

Orbital infections

The child or adolescent who has true orbital disease secondary to sinusitis usually has sudden onset of erythema and swelling about the eye after several days of a viral URI (Fig. 8). Eye pain can precede swelling and often is dramatic. The presence of fever, systemic signs, and toxicity is variable. At least 30% of patients in one series were afebrile at presentation [51]. Orbital infection is suggested by proptosis (with the globe displaced, usually anteriorly and downward), impairment of extraocular eye movements (most often upward gaze), or loss of visual acuity or chemosis (edema of the bulbar conjunctiva). Fortunately, orbital infection is the least common cause of the "swollen eye."

Nearly all orbital infections involve the formation of a subperiosteal abscess. In young children, subperiosteal abscess results from ethmoiditis and

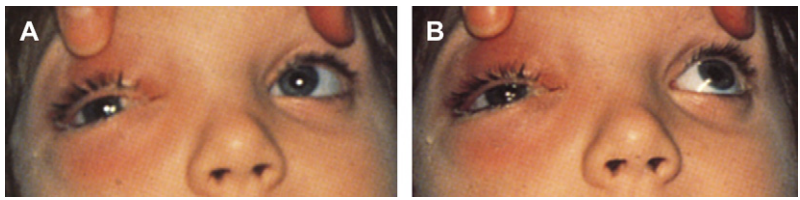

Fig. 8. (*A* and *B*) A 7-year-old boy who has orbital cellulitis. He had a 5-day history of eye pain and progressive swelling of the eyelids, which were markedly erythematous. When his eyelids were retracted, anterior and lateral displacement of the globe and impairment of upward gaze were noted.

ethmoid osteitis. For the adolescent, subperiosteal abscess can be a complication of frontal sinusitis and osteitis. True orbital abscesses are very uncommon [52]. Rarely, orbital cellulitis evolves, without formation of subperiosteal abscess, by direct spread from the ethmoid sinus to the orbit through natural bony dehiscences in the bones that form the medial wall of the orbit [49].

Imaging studies usually are performed if orbital disease is suspected. They help determine whether subperiosteal abscess, orbital abscess, or orbital cellulitis is the cause of the clinical findings (Fig. 9). In the presence of a large, well-defined abscess, complete ophthalmoplegia, or impairment of vision, prompt operative drainage of the paranasal sinuses and the abscess is commonly performed [53–55]. Several studies, however, have reported on the successful drainage of a subperiosteal abscess by endoscopy. This method, performed through an intranasal approach, has avoided an external incision [56,57]. Small abscesses may be managed with intravenous antibiotics alone [58–62]. In many cases, a well-defined abscess is not seen. Instead, inflammatory tissue (a so-called ''phlegmon'') is observed interposed between the lateral border of the ethmoid sinus and the swollen medial rectus muscle. Usually, patients who have these findings also are managed successfully with antimicrobial therapy alone [53,58,59,63]. On occasion, the CT scan can be misleading, suggesting abscess when inflammatory edema is present [50]; accordingly, the clinical course is the ultimate guide to management.

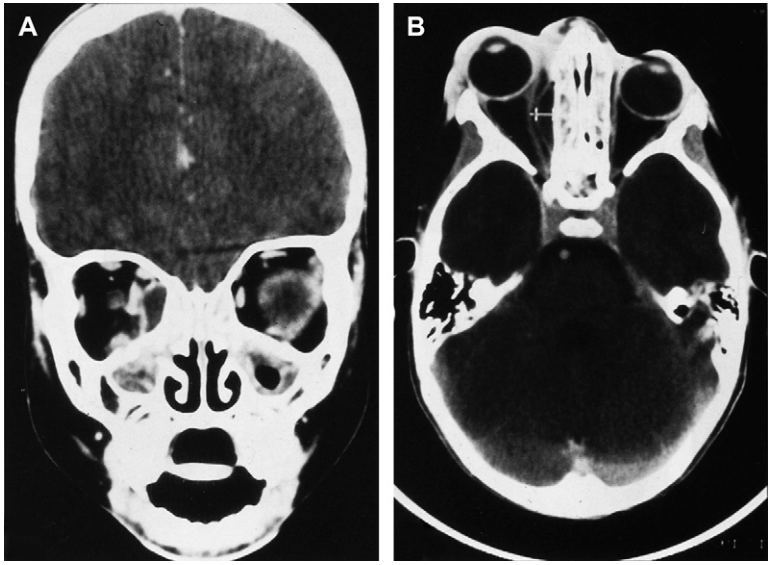

Fig. 9. (*A*) Axial and (*B*). Coronal CT scans show a subperiosteal abscess extending from the left ethmoid sinus.

Empiric antimicrobial therapy should be chosen to provide activity against *S aureus, S pyogenes,* and anaerobic bacteria of the upper respiratory tract (anaerobic cocci, *Bacteroides* spp, *Prevotella* spp, *Fusobacterium* spp, and *Veillonella* spp) in addition to the usual pathogens associated with acute sinusitis (ie, *S pneumoniae, H influenzae,* and *M catarrhalis*) [63,64]. Appropriate selections include cefuroxime (150 mg/kg/d divided into doses every 8 hours) or ampicillin-sulbactam (200 mg/kg/d divided into doses every 6 hours). Clindamycin (40 mg/kg/d divided into doses every 6 hours) or metronidazole (30 to 35 mg/kg/d divided into doses every 8 to 12 hours) can be added if cefuroxime is used and anaerobic infection is likely. If a patient presents with life- or vision-threatening disease, vancomycin may be added to ampicillin/sulbactam for coverage of community-acquired MRSA or penicillin-resistant *S pneumoniae* [51]. If surgery is performed, Gram stain of material drained from the sinuses or the abscess guides consideration of additional drugs or an altered regimen. When final results of culture are available, antibiotic therapy may be changed, if appropriate. Intravenous therapy is maintained until the infected eye appears nearly normal. At that time, oral antibiotic therapy can be substituted to complete a 3-week course of treatment.

References

[1] Abramson DH, Frank CM, Susman M, et al. Presenting signs of retinoblastoma. J Pediatr 1998;132:505–8.
[2] VanDyke RB, Desky AB, Daum RS. Infections of the eye and periorbital structures. Adv Pediatr Infect Dis 1988;3:125–80.
[3] Sirbaugh PE. A case of orbital pseudotumor masquerading as orbital cellulitis in a patient with proptosis and fever. Pediatr Emerg Care 1997;13:337–9.
[4] Greenberg MF, Pollard ZF. The red eye in childhood. Pediatr Clin North Am 2003;50: 105–24.
[5] Lessner A, Stern GA. Preseptal and orbital cellulitis. Infect Dis Clin North Am 1992;6: 933–52.
[6] Chandler JR, Langenbrunner DJ, Stevens ER. The pathogenesis of orbital complications in acute sinusitis. Laryngoscope 1970;80:1414–28.
[7] Shapiro ED, Wald ER, Brozanski BA. Periorbital cellulitis and paranasal sinusitis: a reappraisal. Pediatr Infect Dis 1982;1:91–4.
[8] Block SL, Hedrick J, Tyler R, et al. Increasing bacterial resistance in pediatric acute conjunctivitis (1997–1998). Antimicrob Ag Chemo 2000;44:1650–4.
[9] Bingen E, Cohen R, Jourenkova N, et al. Epidemiologic study of conjunctivitis-otitis syndrome. Pediatr Infect Dis J 2005;24:731–2.
[10] Buznach N, Dagan R, Greenberg D. Clinical and bacterial characteristics of acute bacterial conjunctivitis in children in the antibiotic resistance era. Pediatr Infect Dis J 2005;24:823–8.
[11] Bodor FF. Conjunctivitis-otitis syndrome. Pediatr 1982;69:695–8.
[12] Martin M, Turco JH, Zegans ME, et al. An outbreak of conjunctivitis due to atypical Streptococcus pneumoniae. N Engl J Med 2003;348:1112–21.
[13] Porat N, Greenberg D, Gvon-Lavi N, et al. The important role of nontypeable Streptococcus pneumoniae international clones in acute conjunctivitis. J Infect Dis 2006;194:689–96.
[14] Gigliotti F, Hendley JO, Morgan J, et al. Efficacy of topical antibiotic therapy in acute conjunctivitis in children. J Pediatr 1984;104:623–6.

[15] Leibowitz HM. Antibacterial effectiveness of ciprofloxacin 0.3% ophthalmic solution in the treatment of bacterial conjunctivitis. Am J Ophthalmol 1991;112:29S–33S.
[16] Miller IM, Wittreich J, Vogel R, et al. The safety and efficacy of topical norfloxacin compared with placebo in the treatment of acute, bacterial conjunctivitis. The Norfloxacin placebo ocular study group. Eur J Ophthalmol 1992;2:58–66.
[17] Rose PW, Hamden A, Brueggermann AB, et al. Chloramphenicol treatment for infective conjunctivitis in children in primary care: a randomized double-blind placebo-controlled trial. Lancet 2005;366:37–43.
[18] Sheikh A, Hurwitz B. Topical antibiotics for acute bacterial conjunctivitis: Cochrane systematic review and meta-analysis update. Br J Gen Pract 2005;55:962–4.
[19] Wald ER. Conjunctivitis in infants and children. Pediatr Infect Dis J 1997;16:S7–20.
[20] Ruttum MS, Ogawa G. Adenovirus conjunctivitis mimics preseptal and orbital cellulitis in young children. Pediatr Infect Dis J 1996;15:266–7.
[21] Sullivan JH. Lids and lacrimal apparatus. In: Vaughn D, Asbury T, Riordan-Eva P, editors. General ophthalmology. 13th edition. Norwalk (CT): Appleton & Lange; 1992. p. 28–80.
[22] Lederman C, Miller M. Hordeola and chalazia. Pediatr Rev 1999;20:283–4.
[23] Rhem MN, Wilhelmus KR, Jones DB. Epstein-Barr virus dacryoadenitis. Am J Ophthalmol 2000;129:372–5.
[24] Gungor K, Bekir NA, Namiduru M. Ocular complications associated with brucellosis in an endemic area. Eur J Ophthalmol 2002;12:232–7.
[25] Boruchoff SA, Boruchoff SE. Infections of the lacrimal system. Infect Dis Clin North Am 1992;6:925–32.
[26] Campolattaro BN, Lueder GT, Tychsen L. Spectrum of pediatric dacryocystitis: medical and surgical management of 54 cases. J Pediatr Ophthalmol Strabismus 1997;34:143–53.
[27] Pollard ZF. Treatment of acute dacryocystitis in neonates. J Pediatr Ophthalmol Strabismus 1991;28:341–3.
[28] Hurwitz JJ, Rodgers KJ. Management of acquired dacryocystitis. Can J Ophthalmol 1983; 18:213–6.
[29] Faden HS. Dacryocystitis in children. Clin Pediatr 2006;45:567–9.
[30] Meyer DR, Wobig JL. Acute dacryocystitis caused by *Pasteurella multocida*. Am J Ophthalmol 1990;110:444–5.
[31] Cahill KV, Burns JA. Management of acute dacryocystitis in adults. Ophthal Plast Reconstr Surg 1993;9:38–41.
[32] Powell K. Orbital and periorbital cellulites. Pediatr Rev 1995;16:163–7.
[33] Vayalumkal JV, Jadavji T. Children hospitalized with skin and soft tissue infections. Paediatr Drugs 2006;8:99–111.
[34] Hutcheson KA, Magbalon M. Periocular abscess and cellulitis from Pasteurella multocida in a healthy child. Am J Ophthal 1999;128:514–5.
[35] Velazquez AJ, Goldstein MH, Driebe WT. Preseptal cellulitis caused by Trichophyton (Ringworm). Cornea 2002;21:312–4.
[36] Rajalekshmi PS, Evans SL, Morton CE, et al. Ringworm causing childhood preseptal cellulitis. Ophthal Plast Reconstr Surg 2003;19:244–6.
[37] Jun BK, Shin JC, Woog JJ. Palpebral myiasis. Korean J Ophthalmol 1999;13:138–40.
[38] Caca I, Cakmak SS, Unlu K, et al. Cutaneous anthrax on eyelids. Jpn J Ophthalmol 2004;48: 268–71.
[39] Brannan PA, Kersten RC, Hudak DT, et al. Primary Nocardia brasiliensis of the eyelid. Am J Ophthalmol 2004;138:498–9.
[40] Raina UK, Jain S, Monga S, et al. Tubercular preseptal cellulitis in children—a presenting feature of underlying systemic tuberculosis. Ophthalmology 2004;111:291–6.
[41] Todd JK. Office laboratory diagnosis of skin and soft tissue infections. Pediatr Infect Dis J 1985;4:84–7.
[42] Smith TF, O'Day D, Wright PF. Clinical implication of preseptal (periorbital) cellulitis-Pediatrics 1978;62:1006–9.

[43] Thirumoorthi MB, Asmar BI, Dajani AS. Violaceous discoloration in pneumococcal cellulitis. Pediatrics 1978;62:492–3.
[44] Goldberg F, Berne AS, Oski FA. Differentiation of orbital cellulitis from preseptal cellulitis by computed tomography. Pediatrics 1978;62:1000–5.
[45] Barone SR, Aiuto LT. Periorbital and orbital cellulitius in *Haemophilus influenzae* vaccine era. J Pediatr Ophthalmol Strabismus 1997;34:293–6.
[46] Donahue SP, Schwartz G. Preseptal and orbital cellulitis in childhood—a changing microbiologic spectrum. Ophthalmology 1998;105:1902–6.
[47] Givner LB, Mason EO Jr, Barson WJ, et al. Pneumococcal facial cellulitis in children. Available at: http://www.pediatrics.org/cgi/content/full/106/5/e61. Accessed January 2007.
[48] Schwartz GR, Wright SW. Changing bacteriology of periorbital cellulitis. Ann Emerg Med 1996;28:617–20.
[49] Wald ER. Periorbital and orbital infections. Pediatr Rev 2004;25:312–20.
[50] Skedros DG, Haddad J Jr, Bluestone CD, et al. Subperiosteal orbital abscess in children: diagnosis, microbiology and management. Laryngoscope 1993;103:28–32.
[51] Nageswaran S, Woods CR, Benjamin DK, et al. Orbital cellulitis in children. Pediatr Infect Dis J 2006;25:695–9.
[52] Jain A, Rubin PAD. Orbital cellulitis in childen. Int Ophthalmol Clin 2001;41:71–86.
[53] Sobol SE, Marchand J, Tewfik TL, et al. Orbital complications of sinusitis in children. J Otolaryngol 2002;31:131–6.
[54] Eustis HS, Armstrong DC, Buncie JK, et al. Staging of orbital cellulitis in children: computerized tomography characteristics and treatment guidelines. J Pediatr Ophthalmol Strabismus 1986;23:246–51.
[55] Wong VYM, Duncan NO, Edwards MS. Medical management of orbital infection. Pediatr Infect Dis J 1994;13:1012–3.
[56] Pereira KD, Mitchell RB, Younis RT, et al. Management of medial subperiosteal abscess of the orbit in children—a five year experience. Int J Pediatr Otorhinolaryngol 1997;38:247–54.
[57] Deutsch E, Eilon A, Herron I, et al. Functional endoscopic sinus surgery of orbital subperiosteal abscess in children. Int J Pediatr Otorhinolaryngol 1996;34:181–90.
[58] Rubin SE, Rubin LG, Zito J, et al. Medical management of orbital subperiosteal abscess in children. J Pediatr Ophthalmol Strabismus 1989;26:21–7.
[59] Souliere CR Jr, Antoine GA, Martin M, et al. Selective non-surgical management of subperiosteal abscess of the orbit: computerized tomography and clinical course as indication for surgical drainage. Int J Pediatr Otorhinolaryngol 1990;19:109–19.
[60] Ferguson MP, McNab AA. Current treatment and outcome in orbital cellulitis. Aust N Z J Ophthalmol 1999;27:375–9.
[61] Starkey CR, Steele RW. Medical management of orbital cellulitis. Pediatr Infect Dis J 2001; 20:1002–5.
[62] Greenberg MF, Pollard ZF. Medical treatment of pediatric subperiosteal orbital abscess secondary to sinusitis. J AAPOS 1998;2:351–5.
[63] Harris GJ. Subperiosteal abscess of the orbit age as a factor in the bacteriology and response to treatment. Ophthalmology 1994;101:585–95.
[64] Harris GJ. Subperiosteal abscess of the orbit. Arch Ophthalmol 1983;101:753–4.