Pediatric Orbital and Periocular Infections

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Orbital and periorbital infections are the most common causes of acute orbital inflammation in children. Despite the widespread availability of neuroimaging and advances in antibiotic therapy, these conditions may still result in significant morbidity and mortality. The conditions of orbital cellulitis and preseptal cellulitis may present with similar patterns of eyelid edema and erythema but are two distinct anatomic disease processes with different etiologies. Both conditions have a peak incidence in childhood, but preseptal cellulitis tends to occur in younger children, particularly toddlers [1–5].

Preseptal Cellulitis

Preseptal cellulitis is an infectious process limited to the skin and subcutaneous tissues anterior to the orbital septum. The orbital septum provides a biologic barrier to the advance of infection from the preseptal area into the orbit. Preseptal cellulitis (Fig. 33.1) is the most common type of periocular infection [6] and typically is a less severe process than orbital cellulitis. Preseptal cellulitis, however, can sometimes be associated with sepsis and bacterial meningitis and should not be considered a benign process [7]. Predisposing factors to the development of preseptal cellulitis include local trauma, skin infection like hordeolums, and upper respiratory infection in young children [8]. Less commonly, conjunctivitis, chalazion, and dacryocystitis are implicated as the etiology of the preseptal infection. Complications attributed to preseptal cellulitis include orbital extension of the disease (2.5–17%) [2, 8, 9], skin abscess requiring surgical intervention (8%) [2], eyelid necrosis (1–2%) [10], and intracranial spread of the disease process (2–3%) [2].

Patients with preseptal cellulitis typically present with a short history of eyelid edema and erythema. The eyelid may be warm and tender to palpation. Predisposing factors, such as an insect bite, skin abrasion, hordeolum, chalazion, dacryocystitis, conjunctivitis, or traumatic foreign body, may be identified during the physical exam. Preseptal cellulitis may cause ocular signs such as chemosis and conjunctival erythema. However, proptosis, pupillary changes, and limited motility are signs of orbital involvement and are not included in the constellation of signs of preseptal disease. Fever and elevated white blood cell count are inconsistent features too. The differential diagnosis for preseptal cellulitis includes periocular allergic reactions, bug bites, angioneurotic edema, trauma, renal disease, thyroid eye disease, and early orbital cellulitis without orbital signs.

Preseptal cellulitis is typically treated with antibiotics. In addition, surgical drainage of collections of purulent material is required if an abscess is present. Antibiotic choice is governed by the suspected infective agent(s) and the age of the patient. Neonates and infants are hospitalized for intravenous antibiotics and monitoring. There are varied opinions on the age when outpatient treatment for preseptal cellulitis becomes appropriate. In children, and particularly infants, the immune system is less effective in defending against infection by encapsulated bacteria [11]. An infant's homeostatic mechanisms are also more fragile than those of a child or adult. We do not promote a dog-

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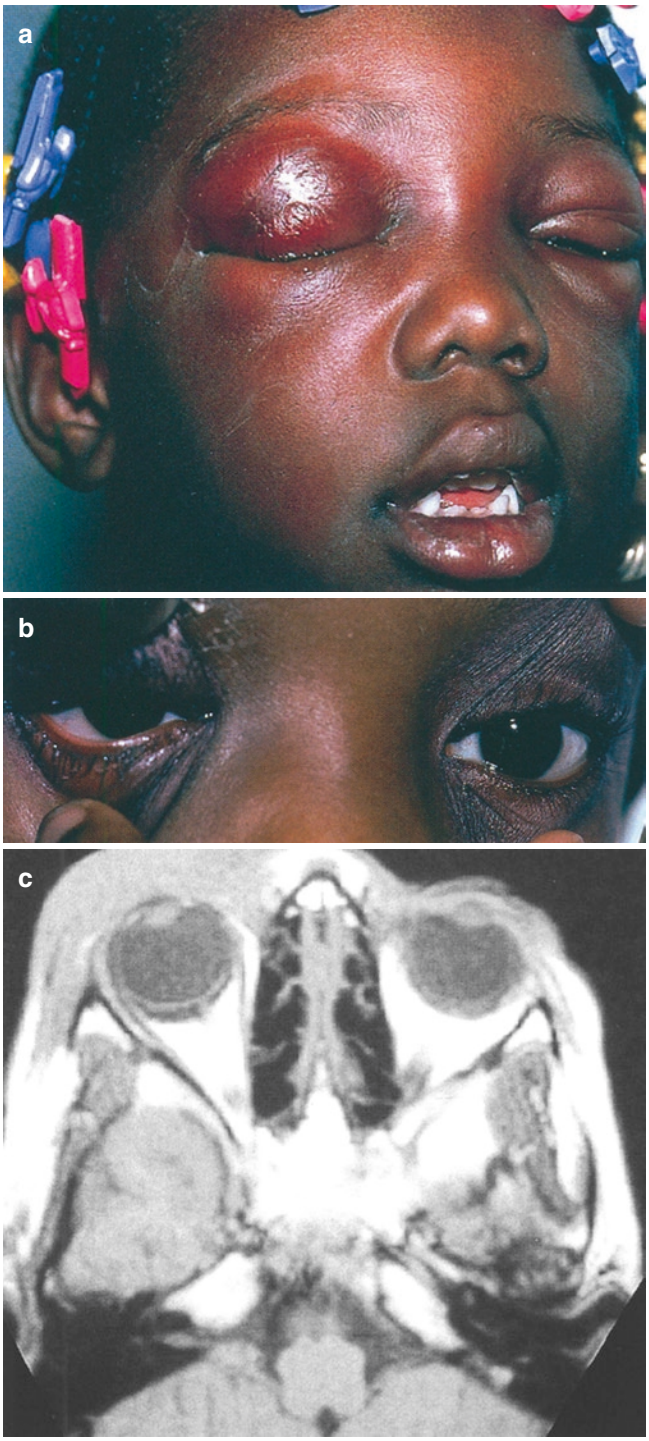

Fig. 33.1 Preseptal cellulitis. (**a**) Clinical appearance of a posttraumatic preseptal cellulitis in a 7-year-old child who scraped her eyelid on the corner of a bed frame. (**b**) Ocular exam on this patient shows absence of conjunctival inflammation, absence of proptosis, and normal motility. (**c**) The MRI scan of this patient shows thickening of the preseptal soft tissue, absence of sinus disease, and no evidence of a foreign body

matic absolute age cutoff for outpatient versus inpatient management of preseptal cellulitis. Rather, we recommend utilization of clinical judgment with a degree of caution. Under the age of 1 year, it is often accepted that a preseptal cellulitis should be managed and monitored on an inpatient basis particularly if the child appears systemically ill or cannot be closely monitored. Between the age of 1 and 5 years, milder cases can be managed with outpatient treatment utilizing appropriate oral antibiotics and close follow-up. Sicker toddlers should be admitted for IV antibiotics. After age 5, it is acceptable to treat stable patients on an oral regimen.

The most common pathogens found in children with preseptal cellulitis are *Staphylococcus aureus* and *Streptococcus* species [7, 8, 12, 13]. Furthermore, methicillin-resistant *S. aureus* has become a more prominent factor in the past 10 years [14]. Other bacteria, like *Streptococcus pneumoniae* and *Haemophilus influenzae*, used to be more prevalent; however, the introduction of the pneumococcal and H. flu vaccines has dramatically reduced the incidence of these infections over the past quarter century [15–18]. Cultures may be obtained by collecting purulent material at the site of the infection, while blood cultures should be obtained in children who are febrile. Culturing the conjunctiva or aspiration of the leading edge of the cellulitis is less rewarding in the yield of positive cultures. Initial selection of antibiotics may be guided by the Gram stain and then modified according to the results of the culture and sensitivities. Specific antibiotic regimens become rapidly outdated, but a suggested regimen is provided below. When available, consultation with a pediatric infectious disease specialist is recommended. When there is a superficial component to the infection, the periorbital skin may also be treated with a topical antibiotic. The preseptal cellulitis should be reevaluated 24–36 h into treatment, at which time improvement should be apparent. Failure to improve or worsening of the disease should initiate a reconsideration of imaging to rule out an abscess, foreign body, or postseptal disease. Antibiotic choices should also be reviewed when culture results are available or be reconsidered with the prevalence of antibiotic resistance that exists today. For routine imaging of a preseptal cellulitis, computed tomography scan (CT) better delineates the bone structure of the orbit, but in cases of a suspected nonmetallic foreign body, magnetic resonance imaging scan (MRI) may be more sensitive.

Surgery is indicated if an abscess or foreign body is identified. These should be drained or removed. The original site of the trauma may be used for surgical access, or an adjacent surgical landmark may be appropriate. Incisions placed in the lid crease in the upper eyelid, the subciliary area in the lower lid, or the lateral canthal angle will leave a cosmetically acceptable scar. After removal of the foreign body and drainage of the purulent material, the area is copiously irrigated with saline or antibiotic solution. The area of the wound or incision may be left open with a drain to prevent reaccumulation of the abscess.

Necrotizing fasciitis (Fig. 33.2) is a more acute and severe infection caused by aerobic or anaerobic microorganisms that is characterized by its rapid spread along and through the soft tissue, causing necrosis of the fascia, overlying skin, and subcutaneous soft tissue. This condition may occur in the eyelids and orbit [19, 20]. Mortality rates for this type of infection range from 30 to 76%. Patients develop systemic toxicity with sepsis, organ failure, respiratory failure, and death. The diagnosis should be considered in any soft tissue infection presenting with signs of systematic toxicity and

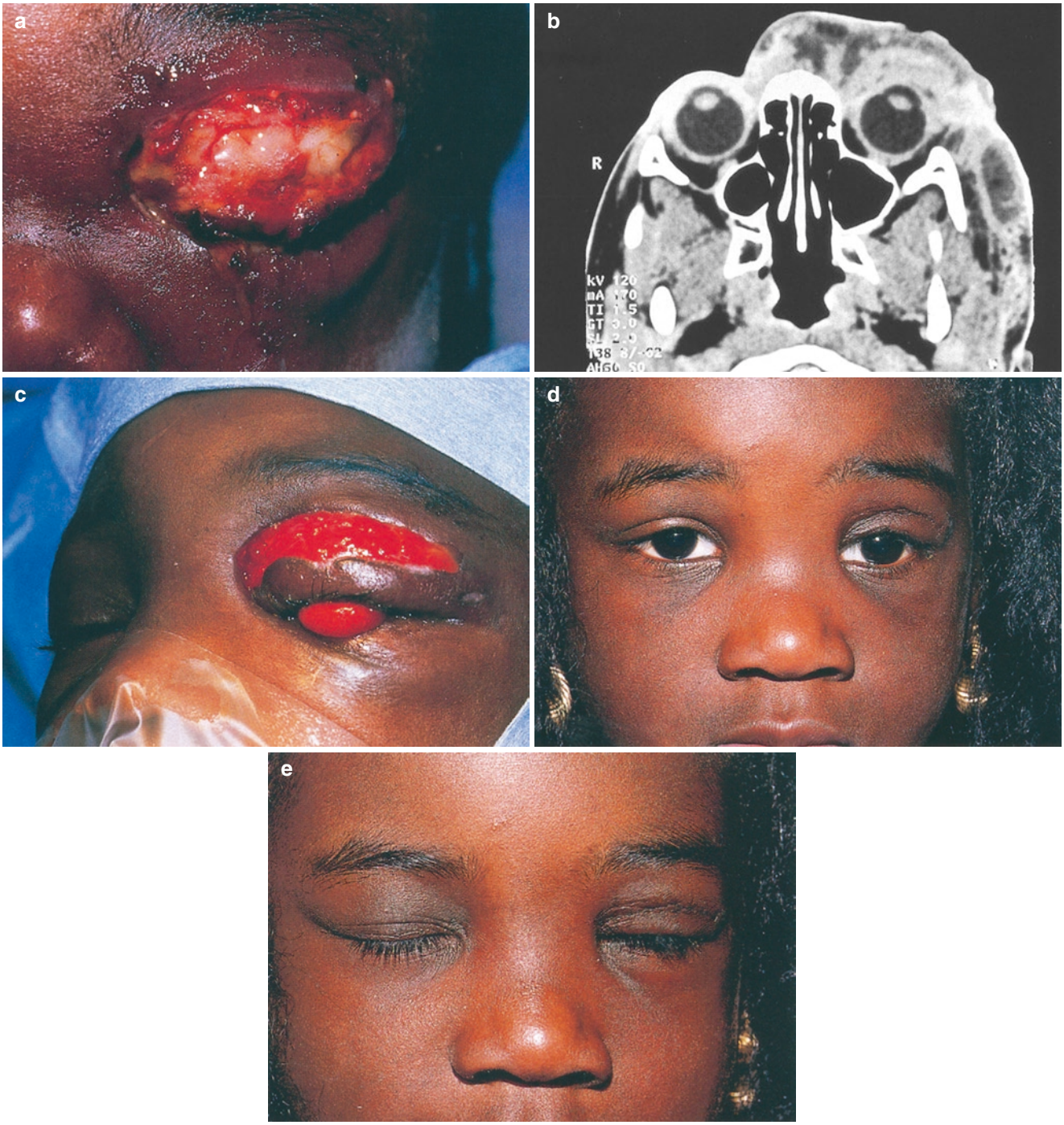

Fig. 33.2 Necrotizing fasciitis. (**a**) This 4-year-old presented with a history of soft tissue trauma to the upper lid with rapidly progressing necrosis of the tissues over the next 2 days. (**b**) The CT scan shows marked soft tissue edema and subcutaneous gas. (**c**) Granulation tissue forming 2 weeks following surgical debridement. Note prolapsed upper cul-de-sac. (**d**) Postoperative appearance 5 months following excision of prolapsed tissue and repair by multiple mattress sutures of 5–0 chromic catgut. No skin grafting was necessary, only spontaneous granulation. (**e**) Downgaze with minimal lid lag

marked edema, because the prognosis is closely linked with early recognition. In children, *Streptococcus* and polymicrobial culture results are reported [20]. Therapy includes broad-spectrum antibiotics (penicillin, aminoglycoside, and metronidazole), early and aggressive surgical debridement, systemic support, and hyperbaric oxygen.

Antibiotic Treatment for Preseptal Cellulitis

Inpatient

Vancomycin 10–15 mg/kg IV Q 8 hours and clindamycin 15–25 mg/kg/day divided Q 8 h.

Outpatient

Trimethoprim/sulfamethoxazole 10 mg/kg/day TMP divided q12 h.

Clindamycin 10–25 mg/kg/day divided Q8 h.

Orbital Cellulitis

Orbital cellulitis is differentiated from preseptal cellulitis by the presence and signs of the postseptal infectious disease process. Orbital cellulitis most commonly arises from spread of contiguous sinus disease but can also arise from traumatic violation of the orbit with implantation of infectious material, trans-septal spread of preseptal cellulitis, or metastatic hematogenous spread of infection to the orbit. The infection may also begin as a dental abscess that spreads to the orbit. Since the orbital process most often arises from spread of infection from the paranasal sinuses, it is helpful to have an ENT consultation involved early with these patients as sinus surgery is an integral part of successful management when surgery is required [21]. Chronic sinusitis is identified as the cause of orbital infection in 75–85% of cases [22]. Conversely, orbital involvement occurs in only 0.5–3% of patients with acute sinusitis [23]. In children, the infected sinus cavities most often seen in association with orbital cellulitis are the ethmoid air cells.

The sinuses and orbit have several anatomic relationships that predispose to the communication of infection and inflammatory processes. Natural dehiscences are often present in the orbital walls, especially the lamina papyracea over the ethmoid sinuses [24]. Bony dehiscences are also reported in the walls of the sphenoid sinus [25]. These disruptions in the sphenoid bone put the optic nerve at risk for septic optic neuritis. The thin wall of the lamina papyracea may also erode in the face of infection, allowing direct communication of the purulent material within the sinus to the subperiosteal space of the orbit. Vascular flow of the valveless orbital veins will allow blood flow to occur from the sinuses to the orbit, another avenue for communication of the infection. The anterior and posterior ethmoidal foramina are additional natural conduits between the sinuses and orbit. All of these features of orbital and sinus anatomy create an avenue for transmission of sinus infection to the orbit.

Definitions of the clinical characteristics of orbital complications of sinusitis were described in a classification system developed by Hubert [26], Smith and Spencer [27], and, more recently, Chandler et al. [6] in the twentieth century. Although the classification system (below) should not be overemphasized as a stepwise progression of the disease process, the description of the clinical characteristics remains a valuable way of defining the severity and location of the infectious process. Orbital imaging is also helpful in staging orbital cellulitis [28]. Nonetheless, it is the clinical exam, disease severity, and progression that are the paramount factors in determining treatment.

Class I (inflammatory edema, Fig. 33.3) is defined by the clinical characteristics of eyelid edema and erythema. This may occur because of congestion of the venous drainage system or transmission of inflammatory mediators from the infected sinuses into the periorbital soft tissue. This stage is sometimes called a preseptal cellulitis, but it is preferable not

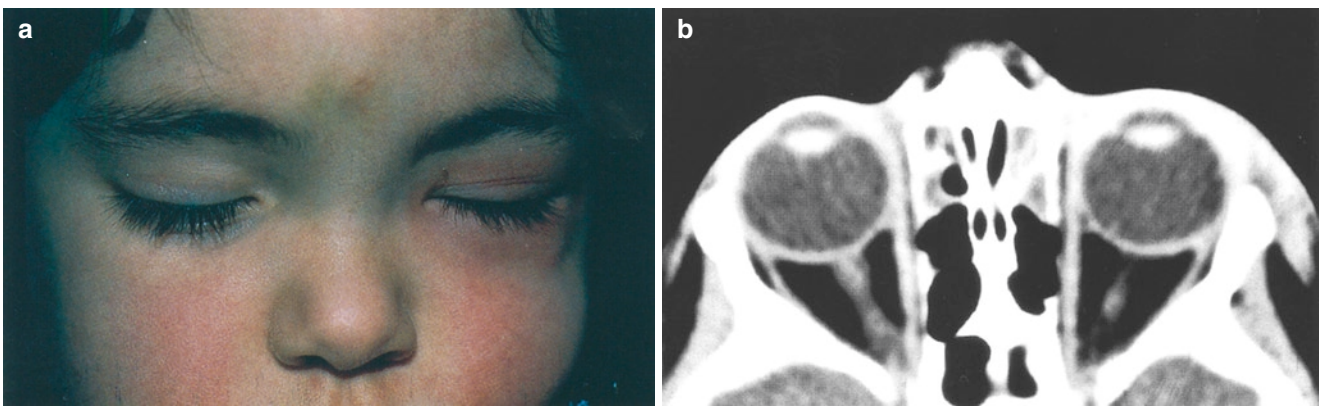

Fig. 33.3 Sinusitis with preseptal inflammation. (**a**) This patient has edema and erythema of the left periocular region. (**b**) The CT scan demonstrates soft tissue edema of the eyelids, opacification of the ethmoid air cells, and normal orbital contents

to use the same term that is used for a preseptal infectious process. In orbital cellulitis, this is an inflammatory response to the sinusitis. Orbital signs of decreased motility, proptosis, or alteration of the pupillary response are absent in the patients who fit the description of class I.

Class II (orbital cellulitis, Fig. 33.4) is differentiated from class I by the presence of eyelid findings and signs of orbital involvement, such as proptosis, restriction of extraocular movements, pupillary defects, and alteration in vision. The diagnosis is confirmed with radiologic evidence of postseptal soft tissue changes. These changes are diffuse soft tissue thickening within the contents of the orbit. Localized collections of infectious material are described in the other classes.

An additional class could be added to the Chandler classes to describe the clinical process of a subperiosteal effusion (Fig. 33.5). This effusion typically develops between the periosteum of the medial orbital and the underlying ethmoid sinuses. The effusion is an inflammatory elevation of the medial periosteum with sterile fluid found between the periosteum and the medial orbital bony wall. By imaging, it is difficult to assess whether this collection is an inflammatory process or whether it will be purulent material [29]. A collection of purulent subperiosteal material is the next classic step in the Chandler classification system (class III subperiosteal abscess, Fig. 33.6). An effusion may resolve when the sinusitis is medically treated, but a subperiosteal abscess is more likely to be resistant to antibiotic treatment and require surgical intervention. Many papers have been written trying to determine what size subperiosteal abscess can be managed medically versus which larger ones require surgery [30–32].

These subsets of patients with subperiosteal fluid collections are challenging to the clinician because the two groups will be similar by clinical appearance and radiologic imaging but will differ in the necessity for surgical drainage. The signs and symptoms of the subperiosteal fluid collections are often similar to class I or class II. At times a subperiosteal

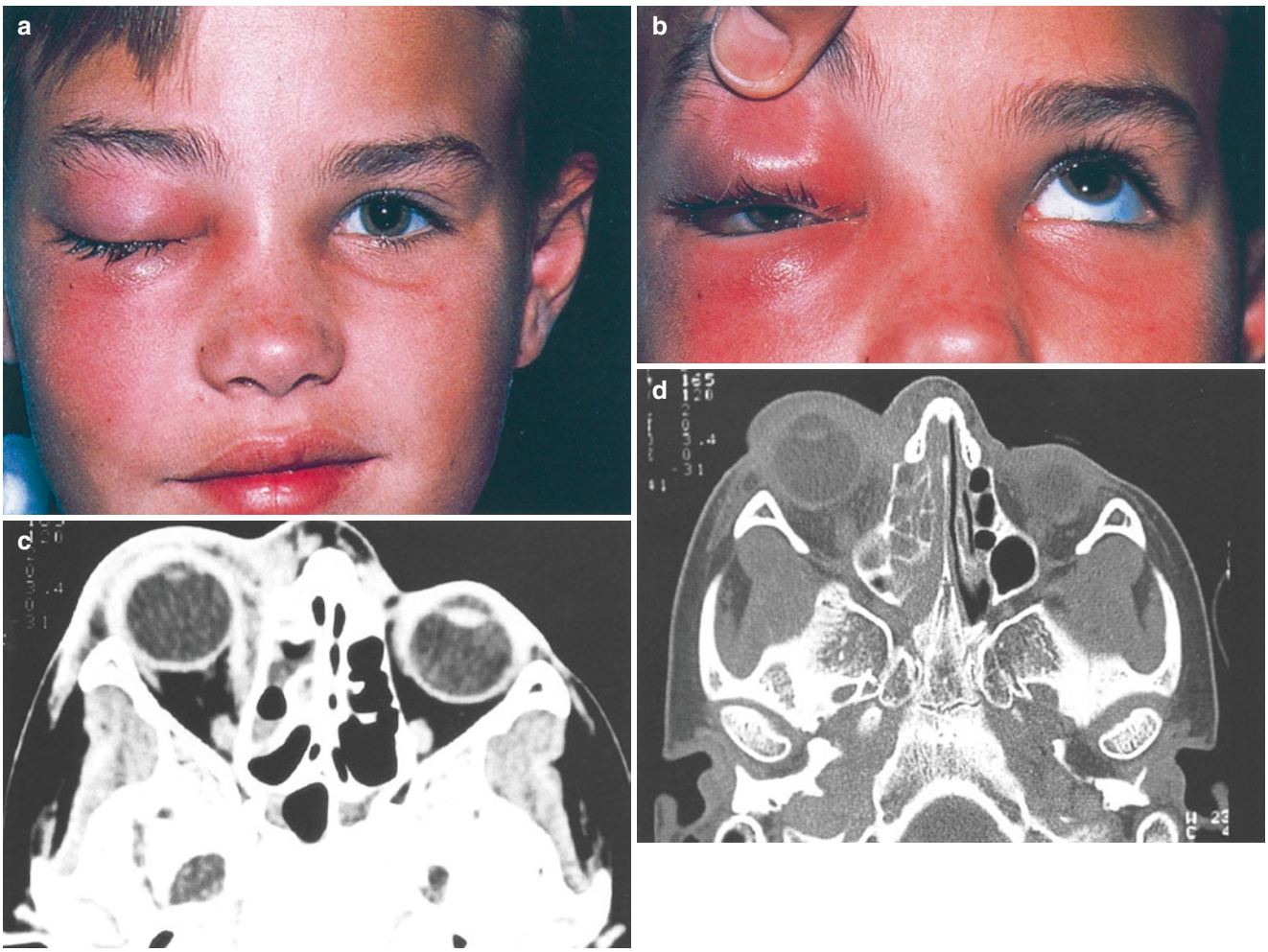

Fig. 33.4 Orbital cellulitis. (**a**) External photograph shows edema and erythema of the right periocular region. (**b**) This patient's attempted upgaze with limitation of motility. (**c**) CT scan shows the soft tissue thickening of the orbital tissue. (**d**) The CT bone windows better demonstrate the extent of the sinus opacification

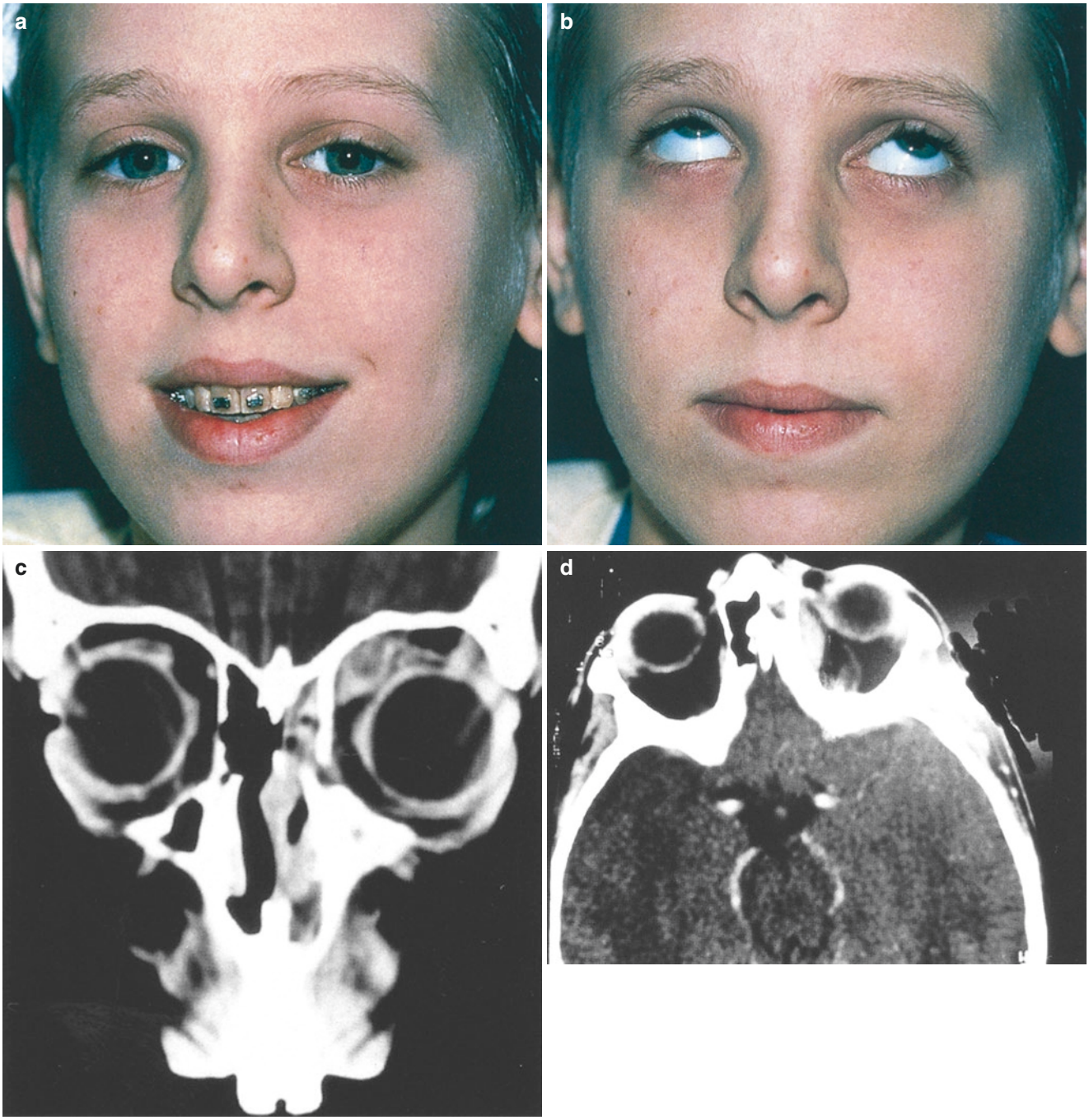

Fig. 33.5 Subperiosteal effusion. (**a**) This 11-year-old patient presented with a complaint of double vision. (**b**) The alteration in motility is seen in upgaze. (**c**) The coronal CT scan demonstrates his left-sided sinusitis and subperiosteal fluid collection. (**d**) Axial CT shows the medial subperiosteal fluid collection. Surgical drainage showed a clear effusion under the periosteum of the medial orbital wall

effusion or subperiosteal abscess will cause a nonaxial proptosis, whereas typically the proptosis caused by diffuse orbital cellulitis will move the globe anteriorly. In the case of a subperiosteal effusion or abscess, the globe is deviated away from the location of the fluid collection. The presence of a subperiosteal fluid collection is recognized only after imaging studies and then becomes a management issue. In a stable patient with good vision and no afferent pupillary defect, it is usually appropriate to initiate a trial of antibiotics rather than moving directly to surgical drainage [33]. Antibiotics alone may clear the sinusitis and an inflammatory effusion may respond without surgical intervention. Patients with decreased vision, afferent pupillary defects, or other deteriorating clinical signs should have urgent drainage

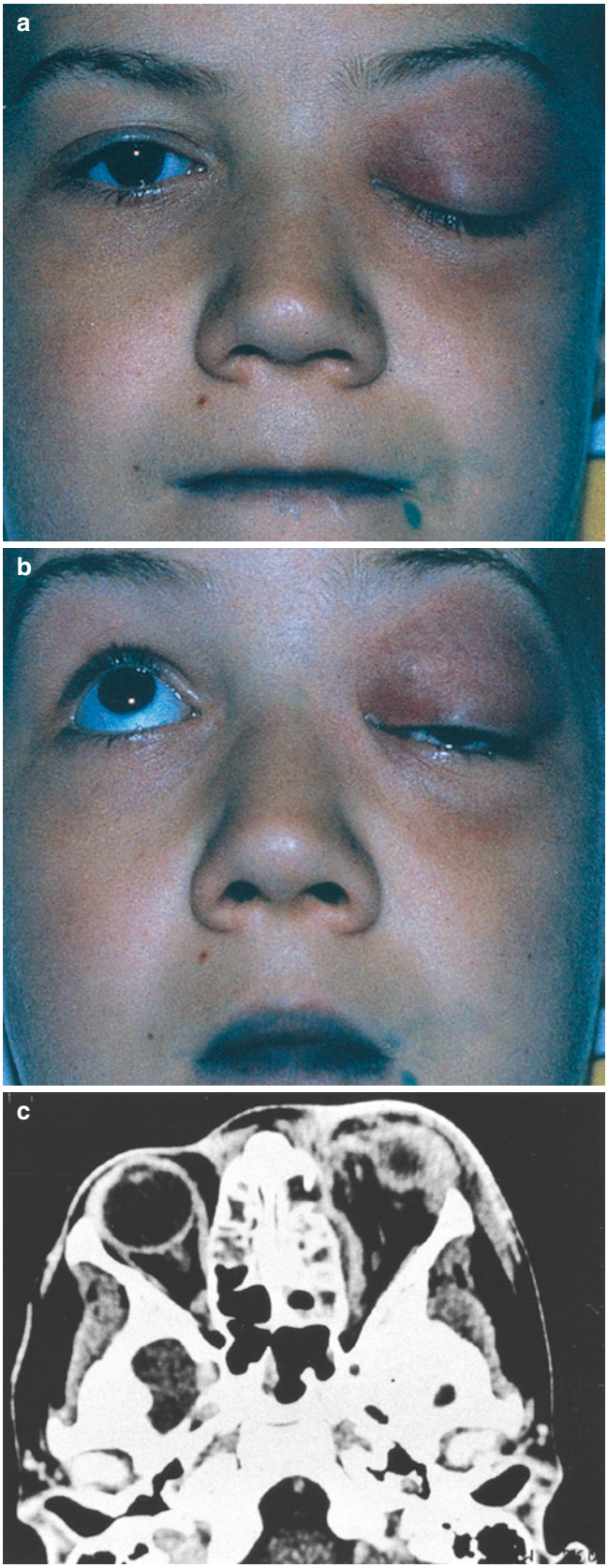

Fig. 33.6 Subperiosteal abscess in orbital cellulitis. (**a**) Photograph in primary gaze demonstrating edema, erythema, and proptosis. (**b**) Photograph in upgaze demonstrating limitation of ocular motility in the left eye. (**c**) CT scan of this patient with ethmoidal sinusitis, a medial subperiosteal abscess, and orbital cellulitis

[30]. At the time of surgery, the subperiosteal fluid may be cultured and smeared to determine whether it is an effusion or purulent fluid.

When the periosteum loses its integrity, or when there is inoculation of the infectious material directly into the orbit, an orbital abscess or collection of purulent material within the soft tissues of the orbit may occur (class IV, orbital abscess, Fig. 33.7). This direct continuity between the purulent material and the orbital tissue puts the orbital structures at higher risk for complications. The septic material and the inflammatory response arc toxic to the orbital tissues. The abscess must be drained, and it is not unusual for successful resolution of an intraorbital abscess to require more than one drainage attempt. However, as noted earlier, combined drainage of sinus infection and the orbital abscess has a lower incidence of reaccumulation of the infection [32]. In the case of an intraconal abscess, it may be necessary to temporarily disinsert an extraocular muscle to access the abscess for drainage. As with any abscess, a pathway for postoperative drainage should be maintained. Sterile rubber bands or Penrose drains may be used for this purpose in the case of an intraorbital abscess.

When the infectious and inflammatory processes follow the venous system posteriorly, a cavernous sinus thrombosis may result (class V). When the infection begins as a unilateral problem, the spread of lid edema and skin discoloration to both sides is a clue to possible extension to the cavernous sinus. Another warning sign is identified when cranial nerve palsies become bilateral or when the motility deficit is identified as a paresis rather than orbital restriction. The physician should be alerted to this when abnormal findings in the extraocular motility are out of proportion with the degree of orbital edema and proptosis. Mental status changes or signs of meningeal irritation will often accompany the cavernous sinus thrombosis. An MRI will be used to diagnose the presence of cavernous sinus thrombosis. *T*2- and proton-weighted images with gadolinium will show high signal luminal narrowing or absence of flow. When a cavernous sinus thrombosis is confirmed, there are relatively few management changes that can be made if the patient has already been adequately treated with antibiotics and all abscesses drained. Heparin therapy has been described, but has not been shown to be helpful in properly controlled studies, and may be precarious in a patient who will need further acute surgical intervention. Ultimately, early suspicion and recognition, with aggressive multidisciplinary treatment, are the keys to managing this rare but devastating complication [34].

Regardless of age, the signs and symptoms of orbital cellulitis mandate hospital admission and intravenous antibiotics as well as dictate the potential need for surgery. The child is admitted and stabilized while intravenous antibiotics are started. As mentioned earlier, antibiotic choices quickly become outdated as the bacteriology of these infections evolve, but suggestions for preliminary choices relevant in 2016 are described below.

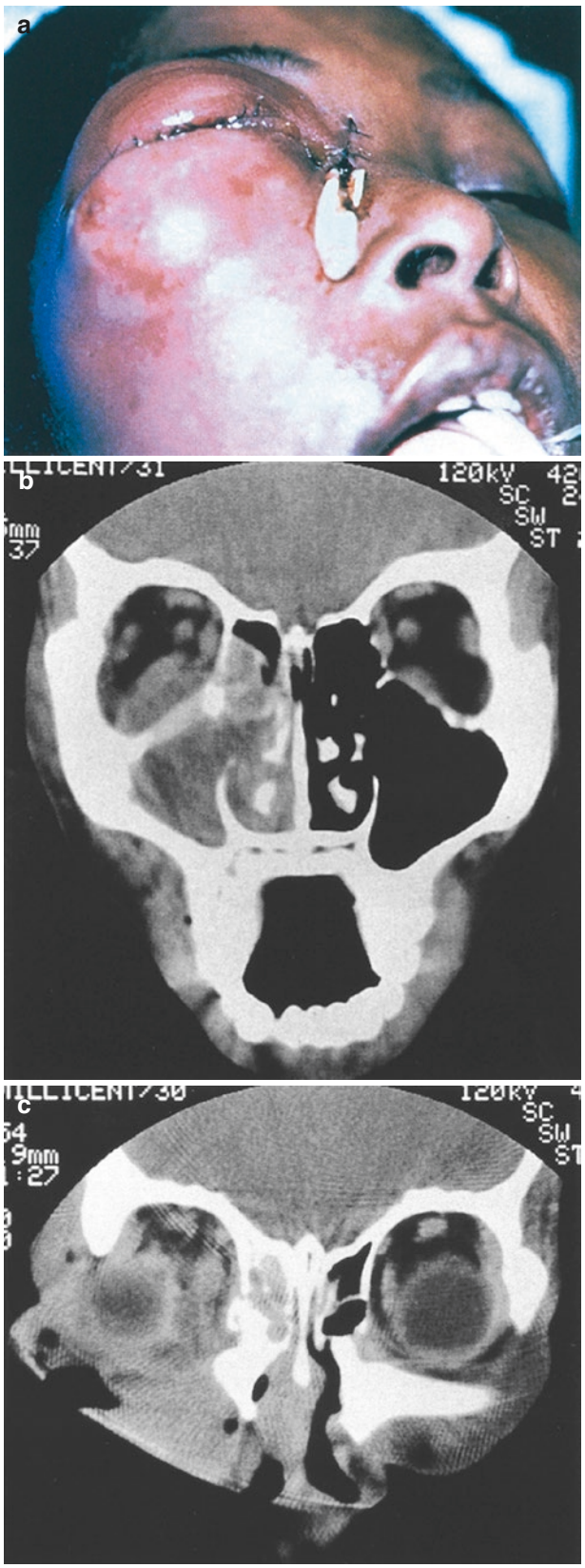

Fig. 33.7 Orbital abscess. (**a**) Intraoperative photo of right orbital abscess. (**b**) CT scan of this patient when the infection was confined to a subperiosteal abscess. (**c**) CT scan 4 days later when the infection has spread to the orbital tissues

Antibiotic choices may be modified according to suspected or cultured bacterial agents. Blood cultures are taken before the antibiotics are started, and other focal lesions, such as conjunctivitis, should be cultured. Screening cultures of normal conjunctiva or the nasopharynx are rarely useful. The organisms responsible for orbital infections are often difficult to determine because of the polymicrobial nature of sinusitis and the abundance of normal bacterial flora on mucosal and skin surfaces. Cultures should be carefully interpreted. Previous antibiotic therapy may also cloud the culture results. In children with orbital cellulitis, the most common organisms isolated by sinus aspirate cultures are *S. aureus*, *Streptococcus* species, and anaerobic species [7, 8, 13, 35]. Particularly in younger children, bacteremia may also be present in cases of orbital cellulitis [23]. Children younger than age 4 have impaired humoral immunity to bacteria with polysaccharide capsules such as *H. influenzae* [7], and bacteremia with these organisms is used to be seen more commonly in this age group. Thankfully, vaccination for *H. influenzae* had a dramatic reduction in these infections.

Once the antibiotics are initiated, the patient is sent for radiologic imaging. Except in certain instances, a CT scan will be requested. That noted, repetition of imaging is unnecessary after the initial scan noting the extent of the infection and treatment is started. Imaging can lag behind the clinical exam, and repeat CT scan, specifically, will expose children to ionizing radiation, which is not entirely innocuous. If cavernous sinus thrombosis, fungal infection, or a vegetative foreign body are anticipated, an MRI scan may be more helpful. In general, the CT scan will adequately image the orbital and sinus processes and better delineate the integrity of the bone.

The results of the imaging are correlated with the patient's signs and symptoms and progress on antibiotic therapy. When the scan of the patient with orbital cellulitis shows sinusitis and/or localized abscess in the orbit in conjunction with a new afferent pupillary defect and significant decreased vision, surgical intervention is indicated. In the child with normal vision and no afferent pupillary defect, close observation is reasonable while medical therapy is initiated. The presence of sinusitis or a subperiosteal elevation in this situation is a softer indication for surgical intervention. Judgment will be used in the face of the patient's total clinical picture over a 24- to 36-h period of observation or immediate intervention with surgery. When medical management is chosen, if there is any deterioration in the clinical signs, or if the patient fails to improve in 24–48 h, surgery is again clearly indicated. The presence of an orbital abscess that is not confined by the periosteum is another immediate indication for surgical intervention.

Early and aggressive use of intravenous antibiotics is often very successful in treating the majority of infections [36]. Children 9 years old and younger usually respond to

antibiotics alone, even if there is a small subperiosteal abscess, as these collections tend to represent simple aerobic bacterial collections [31, 36]. Broad-spectrum coverage is particularly important in teenagers as they typically have polymicrobial infections more similar to adults. Often, patients will show a clinical response in 12–24 h. Again, as long as the patient is clinically stable, there is no significant compromise to visual function and there is no large atypical abscess, the antibiotics should be given an opportunity to work. In addition, steroid therapy can be helpful at reducing inflammation and morbidity once the infection responds to the antibiotics [37]. Steroids are often started after a C-reactive protein level falls below 4 mg/dl [38]. Interestingly, Harris recently looked at the evolution of pathogens causing subperiosteal abscess over the previous 40 years [39]. He noted a change in some of the species of *Staphylococcus* and *Streptococcus*, but generally the same patterns of infection exist. Children under 9 years of age tended to have simple aerobic infections, while older children still had polymicrobial infections. The response rate to antibiotics remained about the same as well. One important difference in the past 10 years, however, has been the increased incidence of methicillin-resistant *Staphylococcus aureus* (MRSA) infection. Thus, as the article notes, the clinical exam supersedes age in determining any need for surgery. Moreover, despite the clinical changes in bacteria from vaccines and antibiotics over time, therapeutic management still remains about the same over the years.

The purpose of surgery when necessary is to drain localized collections of bacteria and purulent material in the orbit and sinus as well as open normal drainage of the sinus into the nasal cavity. In children, this most commonly means an approach to the ethmoid sinuses and medial orbit. The medial orbit can be approached through a lid crease, medial canthal, or transcaruncular incision that also provides access to the ethmoid sinuses (Fig. 33.8). In the older child, endoscopic sinus surgery (FESS) techniques are preferred for sinus drainage. When the maxillary sinus is involved, trans-nasal FESS or a Caldwell–Luc approach may be used for sinus drainage and the inferior orbit drained through a subcillary or lid crease incision in the lower eyelid. In the older child where the frontal sinuses are developed, a superior orbital process is also possible (Fig. 33.9). This may be drained through a lid crease or sub-brow incision. Irrespective of the location of the drainage, an open postoperative drainage site must remain patent to prevent reaccumulation of the abscess. Sterile rubber bands, Penrose drains, or suction drainage may be incorporated.

Other conditions may simulate orbital infectious processes in children, most notably orbital inflammatory disease (see Chap. 34). Orbital inflammatory disease may present with a

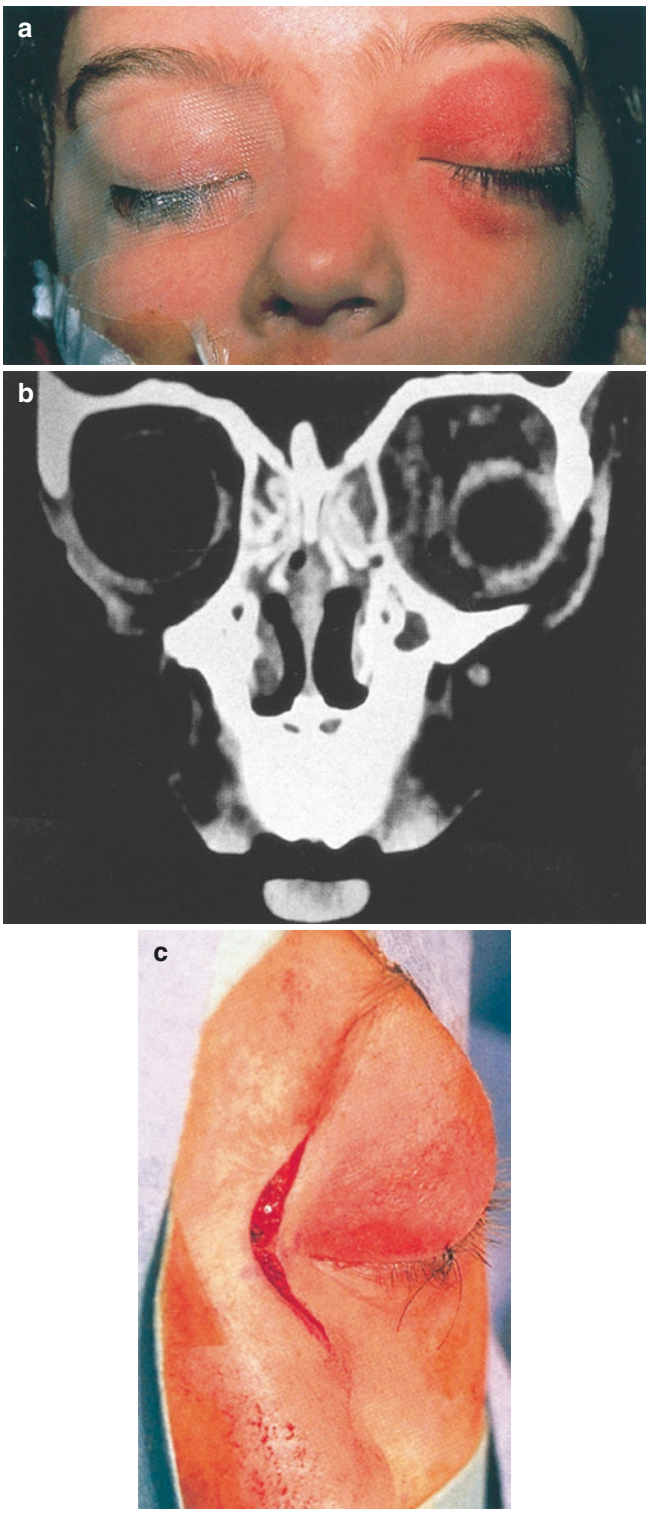

Fig. 33.8 Drainage of ethmoid sinuses and medial orbit. (**a**) A 10-year-old patient with orbital cellulitis, ethmoid sinusitis, and a medial subperiosteal abscess. (**b**) Coronal CT scan demonstrating the radiologic findings; note the medial periosteal elevation over the ethmoid sinuses. (**c**) The abscess is drained through a Lynch incision and an endonasal approach

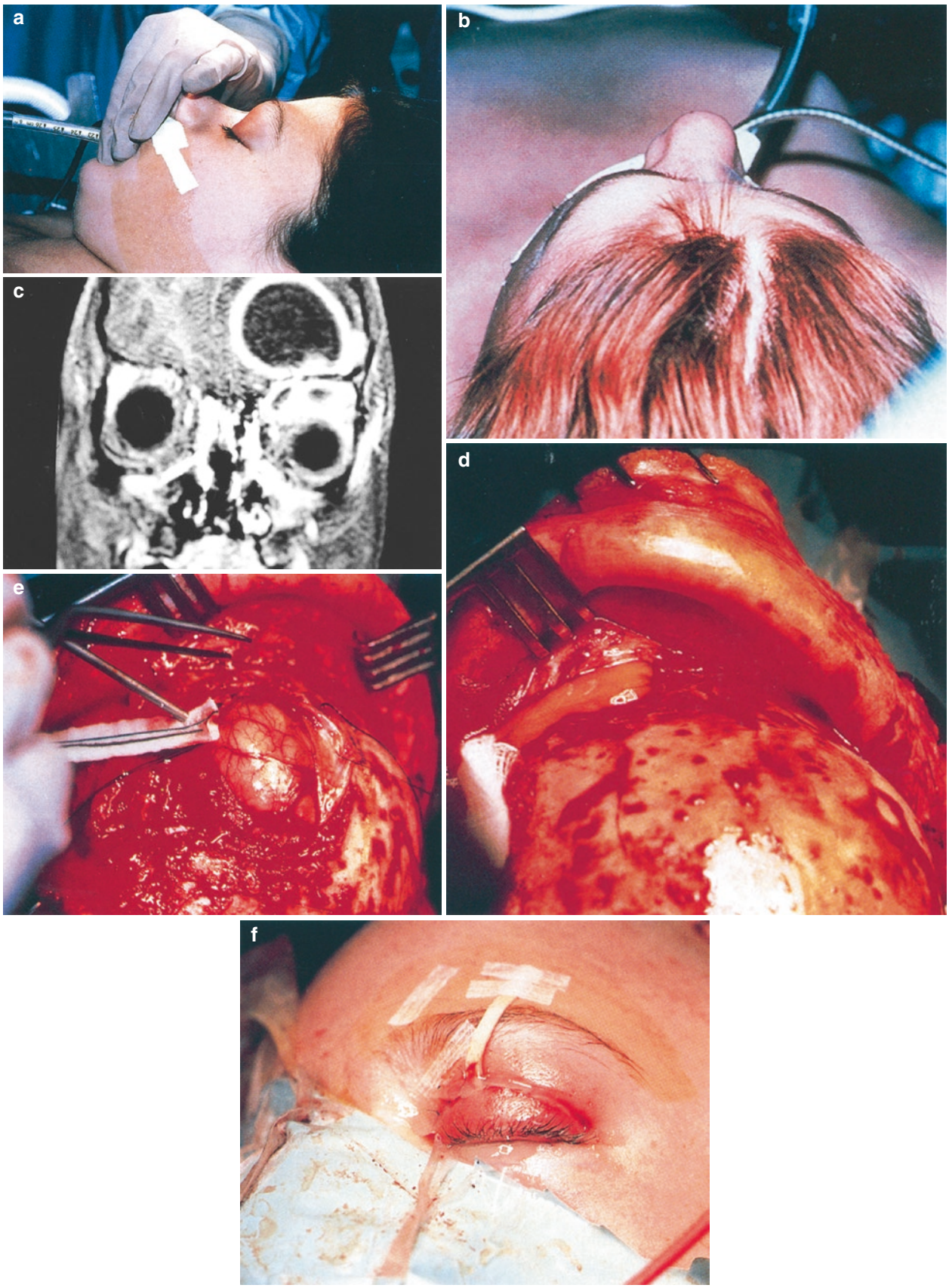

Fig. 33.9 Intraorbital and intracranial abscess. (**a**) This 11-year-old presented with eyelid edema 1 week after being hit in the left eye area with a baseball. (**b**) Proptosis is noted on the patient's left side. (**c**) The MRI scan shows an intraorbital and intracranial collection of fluid. Fractures in the ethmoid and frontal sinuses provided a route for the sinus pathogens to cause an abscess in the superior orbit and in the frontal cortex. (**d**) Drainage of the superior orbital abscess. (**e**) Location of the intracranial abscess. (**f**) External drains in place for the superior orbital abscess

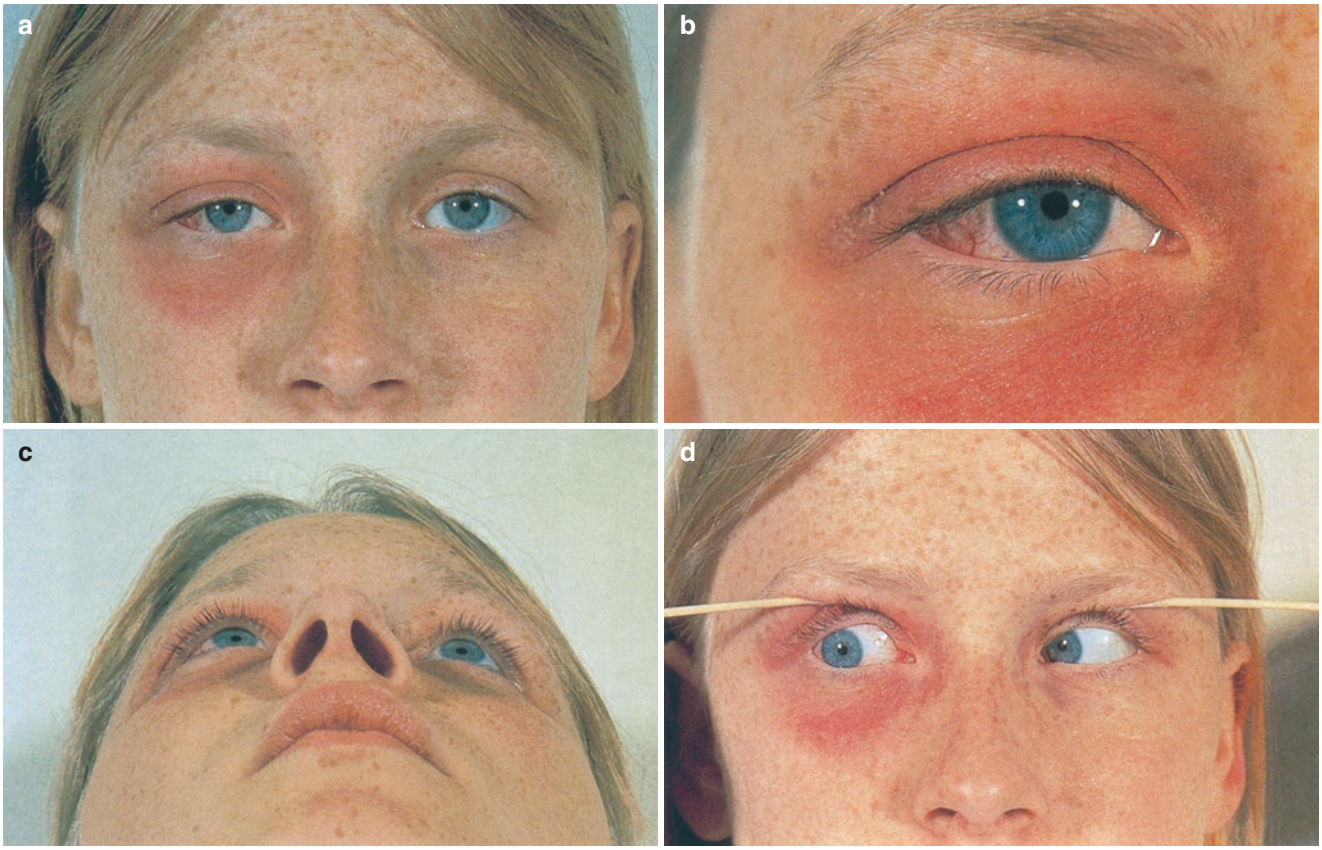

Fig. 33.10 Orbital inflammatory disease. (**a**) Clinical appearance demonstrating pinker hue of periorbital inflammation. (**b**) Early conjunctival chemosis. (**c**) Proptosis. (**d**) Limited mobility on gaze right

similar picture of swelling of the lids, proptosis, erythema of the periocular skin, motility limitations, chemosis, and visual disturbance (Fig. 33.10). Like orbital cellulitis, orbital inflammatory processes are most often unilateral but may be bilateral. There are some subtle differences in the clinical appearance. The color of the periocular inflammation in orbital cellulitis has a redder hue compared to the pinker hue of orbital inflammatory disease. An accompanying iritis may be seen in orbital inflammatory disease but would be atypical of orbital cellulitis. Conjunctival chemosis occurs earlier in orbital inflammatory disease than in orbital cellulitis. Radiologic imaging will help the physician to distinguish orbital cellulitis from orbital inflammatory disease (see Chaps. 32 and 34). The presence of contiguous sinus disease will be a significant feature pointing more to an infectious process. Thickening of the extraocular muscles, enlargement of the lacrimal gland, or a "shaggy" appearance of the orbital fat in the absence of sinus disease will be more typical of an inflammatory process. The clinical picture of the two processes may still be very similar and will sometimes warrant starting IV antibiotics when infection cannot be ruled out. When there are no radiologic indications of infection, and when the condition does not respond to 24 h of IV antibiotics, corticosteroids replace or are added to the antibiotic treatment regimen.

Because of its acute progression and the erythema and thickening of the eyelid skin, rhabdomyosarcoma may also simulate orbital cellulitis. Unlike orbital cellulitis, pain is an unusual feature of rhabdomyosarcoma and may help to distinguish between the two processes. Radiologic imaging will identify bone destruction and invasion with disruption of the architecture of the surrounding structures by the rhabdomyosarcoma. This would be atypical of orbital cellulitis. Biopsy of the sinus or orbital tissue will confirm the diagnosis.

Other conditions that may mimic bacterial orbital cellulitis include carotid cavernous fistula, Wegener's granulomatosis, herpes simplex cellulitis (Fig. 33.11), ruptured dermoid cyst (Fig. 33.12), necrotic retinoblastoma [40], orbital trauma, sickle cell disease with bone infarction (Fig. 33.13), and thyroid eye disease. Imaging, clinical course, and the absence of response to antibiotics are diagnostic features that will provide information to lead the physician to these alternative diagnostic choices.

Despite prompt antibiotic therapy and surgical intervention, orbital infections may lead to serious complications. Intracranial complications such as cavernous sinus thrombosis, subdural abscess, epidural abscess, brain abscess, meningitis, and death have been reported [22, 24, 41]. Decrease or loss of vision may also occur. Mechanisms for this loss of vision include septic optic neuritis, compressive injury to the optic nerve, and thromboembolic lesions to the nerve, retina, or choroid. Permanent orbital scarring may lead to

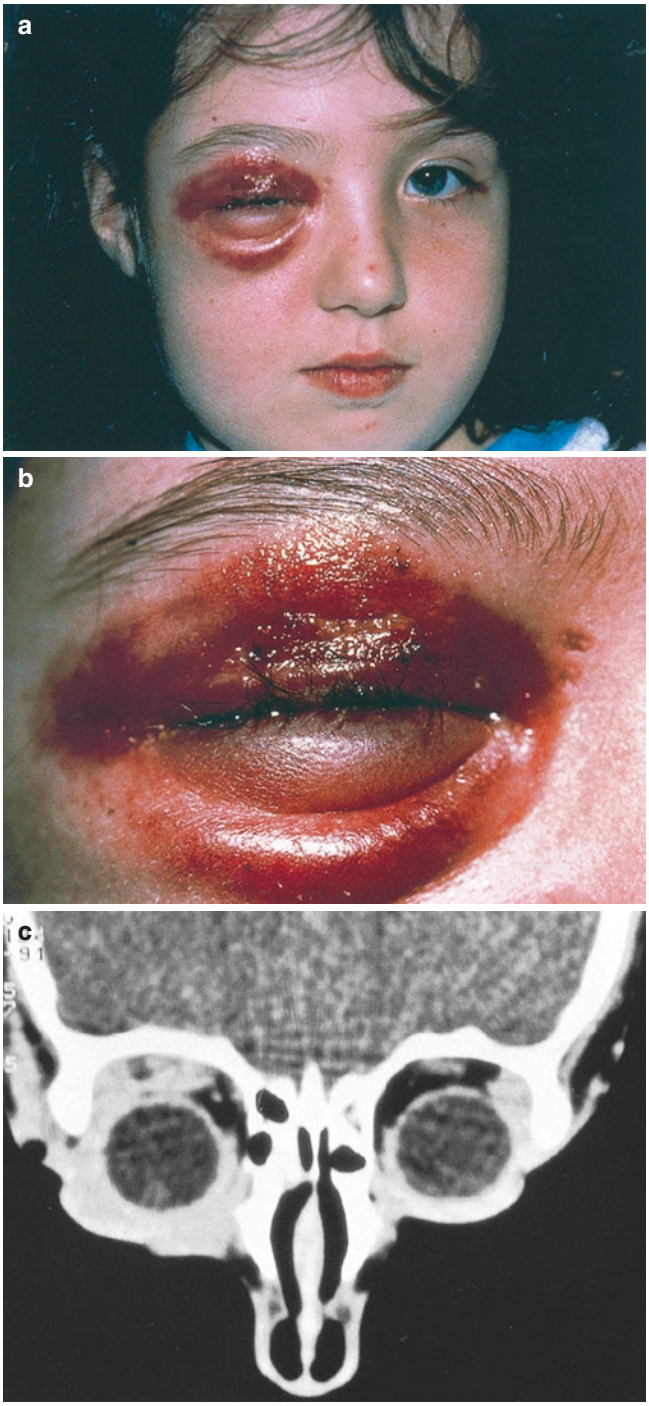

Fig. 33.11 Herpes simplex cellulitis. (**a**) This 12-year-old patient had a mild case of chicken pox and developed a right orbital cellulitis. The process did not respond to intravenous antibiotics, and the tissue culture was positive for herpes simplex I virus. (**b**) The close-up of the eyelid shows the unusual deep red nature of the eyelid erythema. (**c**) The CT scan shows diffuse soft tissue thickening in the orbit with clear sinuses

disruptions in extraocular movement. Scarring of the lids, conjunctiva, and cornea may also occur from the infection or as a result of proptosis and exposure.

Chandler Classification of Orbital Complications in Acute Sinusitis

I. Inflammatory edema
II. Orbital cellulitis
III. Subperiosteal abscess
IV. Orbital abscess
V. Cavernous sinus thrombosis

Antibiotic Treatment for Orbital Cellulitis

Inpatient

Ampicillin/sulbactum 300–600 mg/kg/day divided Q 8 h and clindamycin 15–25 mg/kg/day divided Q 8 h.

Ampicillin/sulbactum 300–600 mg/kg/day divided Q 8 h and vancomycin 10–15 mg/kg IV Q 8 h.

Fungal Infections of the Orbit

Mucormycosis

Mucormycosis is a fungal infection caused by organisms from the genera *Mucor*, *Rhizopus*, and *Absidia.* These are ubiquitous organisms found in air, soil, vegetable matter, bread mold, and fecal material. It is rare for mucormycosis to occur in a normal host [42]. The condition is most commonly seen in diabetic patients with ketoacidosis or in an immunocompromised host.

Mucormycosis affects the orbit by secondary spread from fungal sinusitis. The disease process begins with invasion of the mucosa of the nose, pharynx, or sinuses. Spread occurs by progression along the arterial vascular walls. This leads to occlusion of the vessel and downstream infarction. This vascular thrombosis is responsible for one of the difficulties of treating mucormycosis. The vascular occlusion limits delivery of intravenous antifungal medications to the location of the infection. The infarction also spreads the tissue damage and necrosis beyond the extent of the actual infection.

The typical patient with mucormycosis presents with a history of an underlying medical condition and unilateral orbital inflammation exhibiting cranial nerve palsies that are

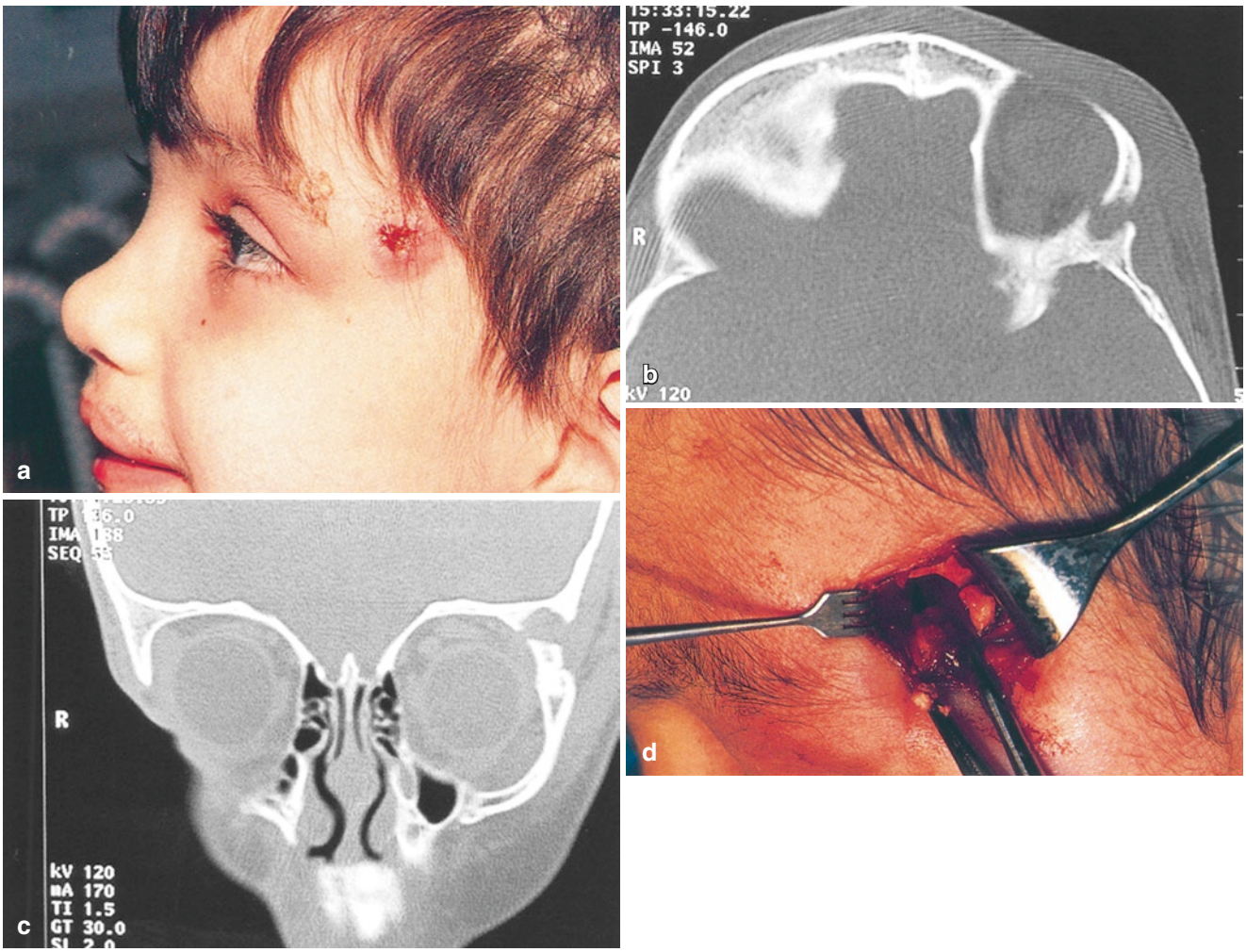

Fig. 33.12 Dermoid misdiagnosed as recurrent orbital cellulitis. (**a**) This 8-year-old had a recurrent episode of orbital cellulitis and purulent draining through a temporal skin fistula following removal of a "sebaceous cyst." (**b**) The axial CT shows soft tissue edema and a natural dehiscence in the bone of the lateral orbital rim. (**c**) The presence of an orbital soft tissue lesion in the area of the bone dehiscence is noted on the coronal CT scan. (**d**) Surgical excision of the "dumbbell" dermoid cyst extending from the orbit to the temporalis fossa

out of proportion with the degree of inflammation. The patient may have severely limited ocular movements, corneal anesthesia, visual loss, and pain. Clues that this represents fungal infection rather than bacterial are the early visual loss and the ophthalmoplegia out of proportion to the level of orbital inflammatory signs. Early visual loss can be caused by central retinal artery occlusion or vascular compromise of the choroidal or ciliary circulation. The nasal and hard palate mucosa may also provide clues to the diagnosis of mucormycosis. A dark, crusted eschar in either location raises the physician's suspicion of a fungal infection, although these signs only occur late in the infection. The diagnosis is confirmed by biopsy of the affected tissue. Large branching nonseptate hyphae will be seen on the hematoxylin and eosin staining.

Orbital and sinus imaging with a CT scan will show sinusitis and orbital soft tissue edema. The CT scan in mucormycosis will sometimes be misleading with a relatively normal appearance, but an MRI scan may be more sensitive in delineating the extent of the sinus mucosal disease.

Treatment for mucormycosis includes surgical debridement of infected and necrotic tissue, systemic amphotericin, amphotericin irrigation, and hyperbaric oxygen. The underlying medical condition that lowered the patient's resistance should be controlled and corrected. For patient in diabetic ketoacidosis, an insulin drip and aggressive approach

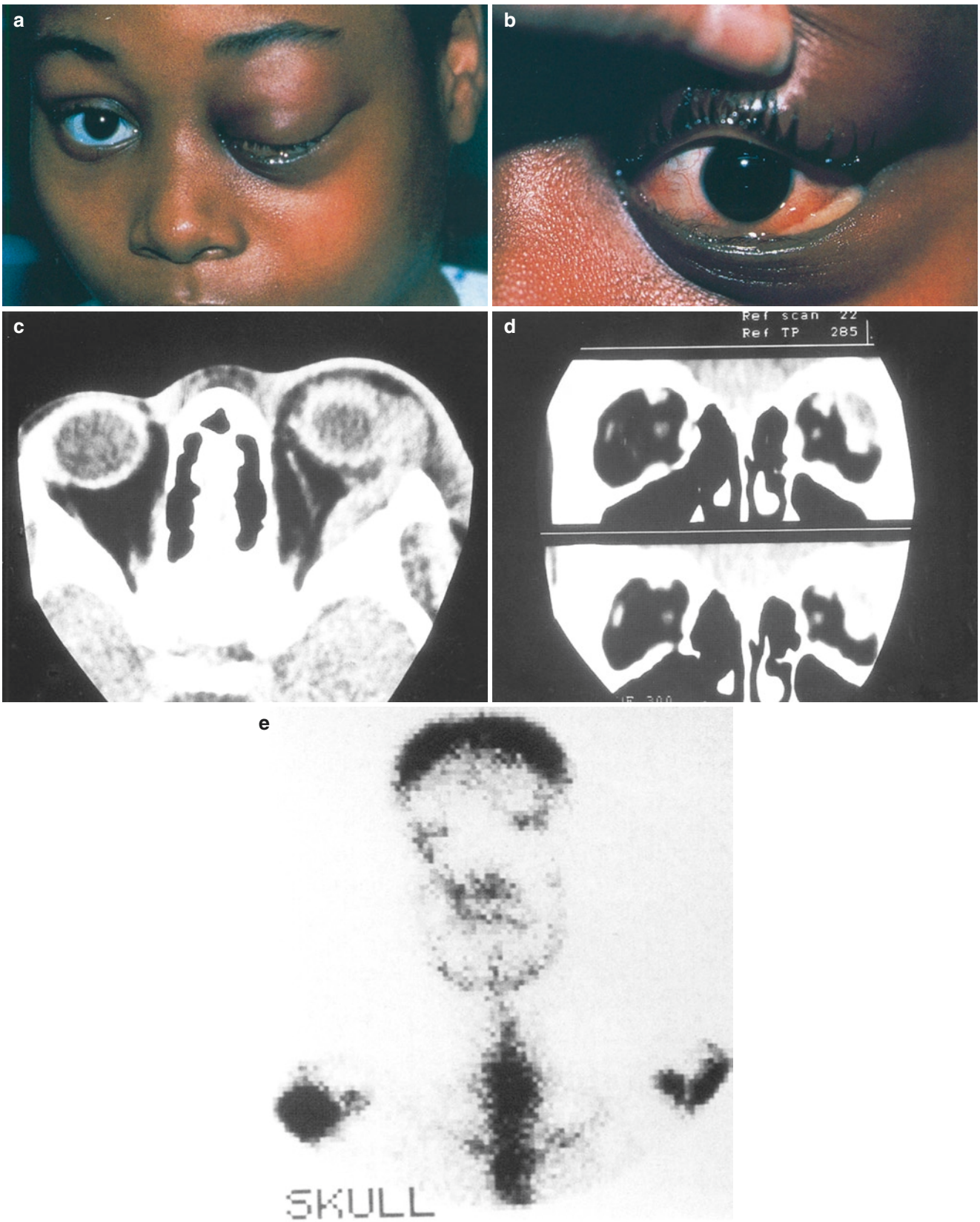

Fig. 33.13 Sickle cell orbital bone infarction simulating orbital cellulitis. (**a**) This 10-year-old patient presented with eyelid edema, eyelid erythema, and pain on ocular movement. (**b**) The conjunctiva of the left eye is chemotic with what appeared to be subconjunctival abscess. (**c**) The axial CT scan showed thickening of the lateral orbital soft tissue and clear sinuses. (**d**) Coronal images show the localized collection of fluid in the superotemporal orbit. Intravenous antibiotics were initiated, but the patient had no clinical response. (**e**) A bone scan was ordered, which identified the bone infarction in the greater wing of the sphenoid

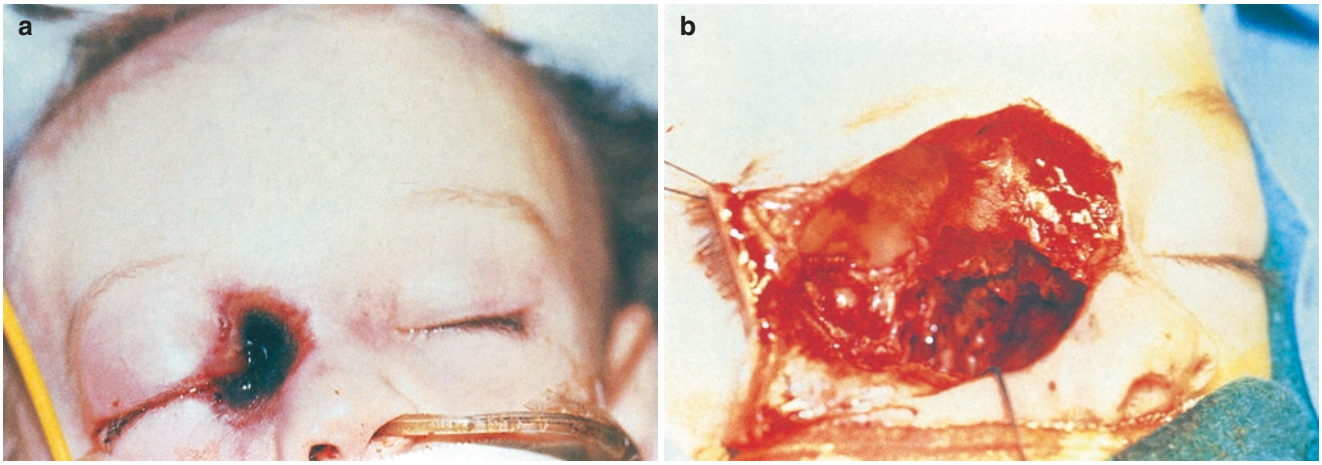

Fig. 33.14 Mucormycosis. (**a**) A 14-month-old child with necrotic medial central soft tissue and lethargy was found to be in diabetic ketoacidosis. Biopsy of the necrotic tissue was positive for fungus. (**b**) Surgical debridement of the affected tissue included exenteration of the orbit

to correcting the metabolic acidosis can be lifesaving. Intravenous amphotericin B in doses up to 1 mg/kg/day can be administered up to a total dose of 2–4 g. Unfortunately, because of the vascular occlusion that is a hallmark of mucormycosis, the necessary surgical resection may be quite large (Fig. 33.14). Aggressive surgical debridement may include exenteration. Again because of limited delivery of intravenous amphotericin to the affected tissues, local irrigation has also been used with some success. The irrigation solution is composed of a 0.25-mg/mL solution of amphotericin B in sterile water. Irrigation of the affected area is performed two times per day [43].

Aspergillosis

Aspergillosis is a fungus of the ascomycetes class. Like *Mucor*, *Aspergillus* is a ubiquitous organism. It can be found colonizing the respiratory and gastrointestinal tracts. In the orbit, aspergillosis can present in at least two clinical scenarios [44]: invasive and infectious. Invasive aspergillosis presents in the immunocompromised host as a sinusitis with rapid progression into the orbit and cranium. Again, like mucormycosis, a necrotizing vasculitis with tissue necrosis is present. This form of the disease is treated with debridement and intravenous amphotericin B. Alternatively, the disease can present in a healthy host. In these cases, a chronic infection of the sinuses occurs with sinus symptoms. There is chronic erosion through anatomic barriers and into the orbit producing orbital signs. The most common signs and symptoms in the orbit are cellulitis, proptosis, ptosis, diplopia, and pain. This form of the disease is also treated with surgical debridement, restoration of normal sinus drainage, and antifungal therapy. Treatment for the immunocompetent host, a more conservative approach to surgical debridement, may still be successful [42].

The diagnosis is confirmed by tissue biopsy showing septate fungal elements on hematoxylin and eosin staining. Multiple biopsies should be taken because of tissue necrosis and better sensitivity to confirm the diagnosis.

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