Surgeon-Related Complications

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Infection

The periocular region has a robust, highly anastomotic vascular supply that allows for a reduced infection rate compared to other surgical sites [1]. The National Nosocomial Infections Surveillance system reported a postoperative infection rate of approximately 2% for all types of surgery [2]. In contrast, studies of common periocular procedures place the infection rate between 0.02% and 0.4% [3–5]. Despite these favorable statistics, serious infections can occur involving atypical mycobacteria, *Staphylococcus aureus*, and group A β-hemolytic *Streptococcus* necrotizing fasciitis.

Periorbital postoperative infections can be grouped into preseptal and orbital cellulitis. Preseptal cellulitis, also known as periorbital cellulitis, is infection that is confined to the eyelid skin and subcutaneous tissues anterior to the orbital septum. On examination, the eyelids are warm, edematous, erythematous, and tender to palpation. The extraocular motility is normal, and there is no proptosis. The most common organisms causing preseptal cellulitis are *Streptococcus pyogenes*, *Staphylococcus aureus*, and *Haemophilus influenzae* type B.

Recent reports suggest an increase in the incidence of cutaneous atypical mycobacteria postoperative infections [6, 7]. Cutaneous atypical mycobacterium infections are often difficult to identify causing a delay in diagnosis with potentially unfavorable outcomes. Onset of infection can range from 1 to 12 weeks postoperatively with the appearance of firm nodules, edema, erythema, and sometimes discharge (Figs. 6.1 and 6.2) [6]. Diagnosis is made by tissue culture and histopathologic evaluation. Treatment consists of systemic antibiotic therapy based upon organism sensitivity and may be combined with surgical debridement. Topical antibiotic treatment may be of benefit. Steroid therapy can prolong the course of infection and

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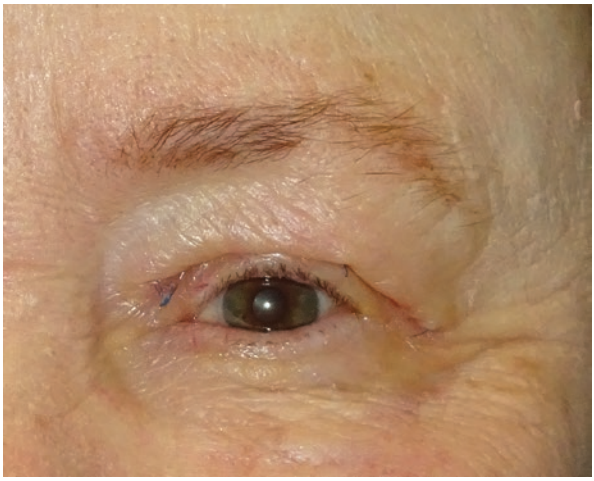

Fig. 6.1 Atypical mycobacteria 1 week post-op: Patient at 7 days postoperative visit following bilateral upper blepharoplasty. Sutures were removed at visit and routine healing was noted bilaterally

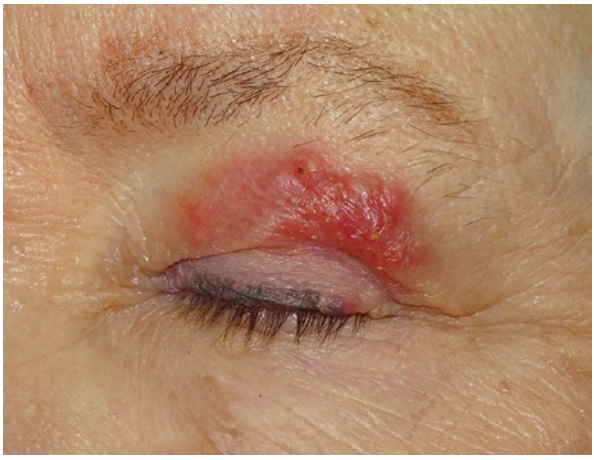

Fig. 6.2 Atypical mycobacteria 2 months post-op: Patient at approximately 60 days postoperative returned with erythema and pustule formation on the left upper eyelid that began 3–4 days prior. Right upper lid was normal. A tissue biopsy was taken which returned a diagnosis of *Mycobacterium chelonae*

should be avoided. Consultation with an infectious disease specialist may be helpful for selecting appropriate antibiotic therapy. Frequent follow-up is required to assess the treatment progress. The duration of treatment is typically greater than 5 weeks with a range of 4 to 24 weeks (Fig. 6.3) [6, 8].

Orbital cellulitis occurs when the infectious process involves the tissues posterior to the orbital septum. The clinical presentation differs from preseptal cellulitis by the additional symptoms of increased pain, proptosis, restriction of ocular motility, pupillary defects, dyschromatopsia, and loss of vision. Alterations in globe position, proptosis (exophthalmos), pupillary defects, and significantly decreased vision should not be seen if the process is isolated to the preseptal area. The most common causative organisms of orbital cellulitis are the same as those involved in preseptal

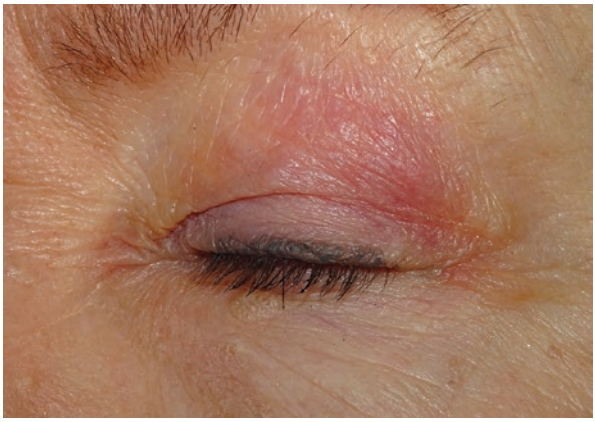

Fig. 6.3 Atypical mycobacteria 2 months post-treatment – 4 months post-op: Patient at 120 days postoperative and 60 days post-oral and topical antibiotic treatment for mycobacterium infection of the left upper lid. Patient was placed on oral clarithromycin and topical tobramycin ophthalmic ointment. Tissue texture and erythema gradually improved with appropriate treatment

cellulitis: *Streptococcus pyogenes*, *Staphylococcus aureus*, and *Haemophilus influenzae* type B.

Preseptal cellulitis is initially managed with oral antibiotics and clinical monitoring. Orbital cellulitis management is more aggressive, and early recognition and treatment is paramount to prevent further complications, such as subperiosteal abscess, orbital abscess, cavernous sinus thrombosis, optic nerve compression, meningitis, panophthalmitis, brain abscess, or vision loss. In cases of orbital cellulitis, the patient is admitted to the hospital for close observation, computed tomography (CT) of orbits and the brain is performed, and blood cultures and intravenous (IV) broad-spectrum antibiotics are started immediately. No other testing should delay treatment with the IV antibiotics. The patient should be monitored for clinical progression or improvement with serial visual acuity, pupillary testing, and confrontation visual fields. In the absence of noticeable improvement within 24–48 hours, CT should be repeated, and addition or alteration of antibiotics is considered. Radiologic identification of an orbital abscess, particularly in an adult, typically warrants surgical intervention.

Surgical intervention is indicated when there is inadequate improvement with antibiotics and/or evidence of an orbital abscess, progressive visual loss, and visual field constriction. There are primarily two types of interventions – orbital decompression and orbitotomy with exploration and drainage. Surgical approaches for drainage of orbital abscess include opening the upper eyelid blepharoplasty incision to reach the affected area, the Kronlein-Burke approach (lateral orbital wall orbitotomy), an inferior transconjunctival incision following the pathway of the lower eyelid blepharoplasty incision, or a transantral Caldwell-Luc decompression

(medial and inferior wall orbitotomy). The medial orbit can also be accessed through a trans-nasal endoscopic approach.

Orbital Hemorrhage

Orbital hemorrhage is one of the most feared complications of orbital and periorbital surgery. Orbital hemorrhage is the most common cause of postoperative permanent vision loss, and it usually occurs within the first 24 hours after surgery (Fig. 6.4). The mechanism of permanent vision loss from orbital hemorrhage is believed to be elevated intraocular (IOP) and intraorbital pressure (OP) caused by hematoma. Orbital compartment syndrome (OCS) describes a condition where there is an increase in intraorbital pressure within the confined orbital volume. When the intraorbital pressure exceeds the arterial pressure, optic nerve or choroidal ischemia may lead to irreversible vision loss. Risk factors for orbital hemorrhage include thyroid associated orbitopathy, blood dyscrasias, hypertension, atherosclerosis, vascular disease, and anticoagulation [9]. Careful assessment of the patient's risk factors for hemorrhaging should be performed prior to performing periorbital surgery. Any patient that complains of extreme pain, asymmetric swelling, proptosis, dimming or loss of vision postoperatively might have an orbital hemorrhage and must urgently be evaluated and treated. A complete ophthalmologic examination, including visual acuity, pupil assessment, intraocular pressure, and dilated fundus exam, should be performed but should delay treatment if there is an obvious collection of blood. Diagnosis is primarily made by clinical examination; however, orbital CT may be a useful adjunct.

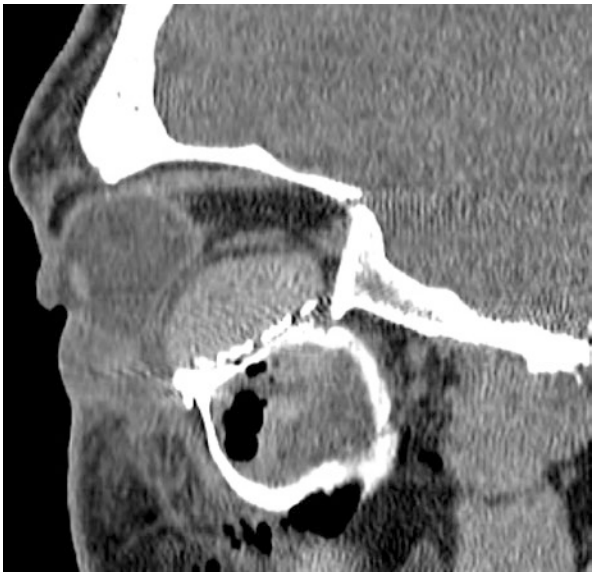

Fig. 6.4 Sagittal CT image showing a large orbital hemorrhage 12 hours following orbital floor fracture repair. The titanium implant can be seen inferior to the hemorrhage. Orbital compartment syndrome was diagnosed clinically in this case based upon the deformation of the globe and nerve on CT, a complete APD, increased IOP, globally decreased ocular motility, orbital pain, and no light perception (NLP) vision

Intraoperative hemostasis is crucial in prevention of orbital hemorrhage. In the postoperative period, one may consider the use of antiemetics to prevent hemorrhage secondary to valsalva. Medical management may include the use of systemic corticosteroids to reduce orbital and periorbital tissue edema. If intraocular pressure is elevated, topical or systemic medications to control intraocular pressure may be used to temporarily protect the optic nerve. Treatment of elevated intraocular pressure is only a temporizing measure to decrease the incidence of optic nerve insult. However, decreasing the intraocular pressure with combinant increased orbital pressure may further diminish intraocular blood flow. In cases where vision is intact and the hemorrhage is felt to be stable, progression can be followed with monitoring of the pupils and color plates, exophthalmometry, and serial Humphrey or Goldmann visual field testing. Serial orbital CTs can also be used to monitor progression; however, there are radiation risks with repeated exposures. There are also disposable compartment pressure measuring devices that use a standard 18-gauge needle and can provide objective orbital pressure measurements [10].

Definitive management of a progressive orbital hemorrhage causing orbital compartment syndrome is surgical intervention. The periorbital incision can be opened and explored, cauterizing or ligating any potential sources of bleeding. Any visualized clots are removed and compartmentalized blood evacuated. If the patient's presenting condition is severe, or progresses, then a lateral canthotomy and cantholysis is performed. When this does not sufficiently reduce the vision threatening symptoms, orbital decompression may be performed. Decompression can involve the canthal cutdown technique, where the orbital septum is opened at the site of the canthotomy incision and blunt dissection is used to disrupt the various orbital compartment septae [11]. The septum is incised at the 7–8 o'clock position in the right eye and 4–5 o'clock position in the left eye as these areas are devoid of ocular muscles and nerves. The inferolateral orbit can also be explored through the cantholysis opening to release loculated compartments of heme within the orbital septae [12]. If further decompression is required, the orbital floor can be infractured with a freer elevator. In the majority of cases, canthotomy and cantholysis, with canthal cutdown, are adequate to reduce the orbital pressure and prevent further optic nerve compromise. It is possible to reverse visual loss caused by orbital hemorrhage with immediate and aggressive surgical intervention. Therefore, recognizing the signs and symptoms of orbital hemorrhage and prompt intervention are instrumental in preventing permanent vision loss.

Globe Perforation

Globe perforation, or rupture, can occur during any periocular procedure. The globe is at risk for perforation during injection of local anesthetics, incision, dissection, cautery, laser usage, and suturing (Fig. 6.5). Globe perforation is an ophthalmic emergency and can lead to permanent vision loss. Perforation risks can be decreased by the use of plastic or metallic corneoscleral protective shields, placed prior to

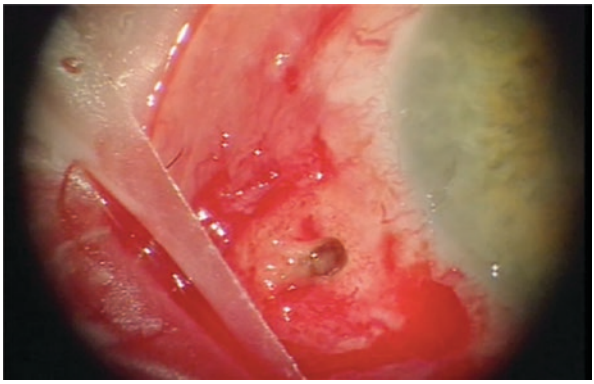

Fig. 6.5 Full-thickness penetration of globe, possibly from overaggressive removal of nasal fat pad with monopolar cautery. The patient went on to develop endophthalmitis resulting in no light perception vision (NLP)

surgery. A topical ocular anesthetic should be placed on the eye prior to insertion. Anesthetic should be applied approximately every 45 minutes or when the patient complains of ocular discomfort. If using laser for incision or dissection, nonreflective metallic shields should be used.

The placement of a corneoscleral shield at the start of surgery and proper injection and surgical technique can help decrease the chances of globe perforation, but the shield will only protect the area it covers. It is still possible for uncovered portions of the sclera to be perforated. The conjunctiva can also be lacerated without perforation. Seidel testing can be performed to evaluate for a leak of aqueous humor or vitreous from the wound to rule out perforation. However, puncture wounds can be self-sealing. A dilated funduscopic evaluation should be performed in the event of any suspected perforation incident.

Globe perforation or rupture is an ophthalmic emergency. The surgical procedure should be halted. There should be no manipulation of the eye. Broad-spectrum antibiotics should be given topically and intravenously, and an eye shield placed over the eye. An ophthalmologic evaluation must be performed immediately for evaluation and emergent repair if required.

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