Anterior Segment Complications of Herpes Zoster Ophthalmicus

Stephen C. Kaufman, MD, PhD

Topic: The clinical features and management strategies for varicella-zoster virus (VZV) infections of the cornea, lids, and adnexa.

Clinical Relevance: Herpes zoster ophthalmicus (HZO) can result in a myriad of chronic and recurrent complications that may be sight threatening. Surgical intervention is the mainstay of treatment, and advancements in this area may lessen the complications of HZO if correctly implemented.

Methods: Literature review of pertinent topics, authors, and journals utilizing the National Institutes of Health PubMed service.

Results: A higher rate of treatment success for VZV-related complications was obtained when any preexisting ocular inflammation, increased intraocular pressure, lagophthalmos, dry eye, exposure, or neurotrophic keratitis was treated and under control before attempting ocular surgery.

Conclusion: Options are available to manage ophthalmic complications of HZO and reduce the risk of treatment failure. *Ophthalmology 2008;115:S24–S32 © 2008 by the American Academy of Ophthalmology.*

It is estimated that the lifetime risk of developing a herpes zoster (HZ) viral infection is 10% to 20%.[1] The reactivation of the varicella-zoster virus (VZV) originates from the sensory ganglia. When the infection involves the first division of the trigeminal nerve (ophthalmic division), this disorder is termed herpes zoster ophthalmicus (HZO). Of the 3 divisions of the trigeminal nerve, the most commonly involved in HZ is the first.[2] In fact, the first division of the trigeminal nerve may be involved 20 times more frequently in an HZ infection than the second or third division.

When the VZV reactivates, viral replication takes place in the sensory ganglia and spreads to the corresponding dermatome innervated by that ganglion. The precise factors that influence reactivation are unclear. However, as the virus replicates the viral DNA and proteins are assembled, and the virus obtains its outer envelope as it is released from the cell.[3] Secondary reinfection by VZV has been reported and confirmed by transient elevation of varicella-specific antibodies.[4] Histologic specimens from eyes affected by HZO demonstrated inflammation in an assortment of different ocular tissues. Additionally, inflammation may spread locally and result in neuritis and vasculitis.[5] Herpes zoster ophthalmicus can result in a variety of ophthalmic sequelae, which may be chronic and recurrent in nature. Despite advances in therapy, HZO-related complications are lessened but not eliminated.[1] The complications from HZO can be sight threatening. This article discusses serious potential complications of VZV infections of the cornea, lids, and adnexa and their treatment.

From the Department of Ophthalmology, University of Minnesota, Minneapolis, Minnesota.

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Correspondence to Stephen C. Kaufman, MD, PhD, Department of Ophthalmology, University of Minnesota, 420 Delaware Street SE, MMC-493, Minneapolis, MN 55455. E-mail: corneamd2000@yahoo.com.

Lids and Ocular Adnexa

Although the rash of varicella does not typically result in scarring, HZ often produces cicatricial skin changes. Because the skin of the forehead and lids is thin, the secondary scarring may be more apparent in these areas. Early in the course of disease, the eyelids may become hyperemic and edematous. If the swelling of the eyelid is significant, ptosis may result. Conversely, later in the course of the disease, loss of eyelid mobility through cicatricial changes of the skin or a palsy of the orbicularis oris muscle may result in lagophthalmos (Fig 1). Also, the affected area may become hypersensitive to any stimulus, which makes any manipulation of the lids extremely painful. This hypersensitivity of the lids and adnexa can make routine treatment of the HZO complications very painful; however, exposure must be treated to avoid corneal scarring and ulceration.

Lagophthalmos

Lagophthalmos comes from the Greek *lagos,* meaning hare. It was once believed that rabbits slept with their eyes open. Individuals with an impaired ability to close their eyelids fully are said to have lagophthalmos. Lagophthalmos can result in severe complications after an HZO infection; however, even a limited degree of lagophthalmos may result in epitheliopathy and more serious corneal damage resulting from increased tear evaporation, poor tear film, surface drying, and corneal surface breakdown.

After an episode of HZO, patients may complain of ocular irritation, a foreign body sensation, or stinging and

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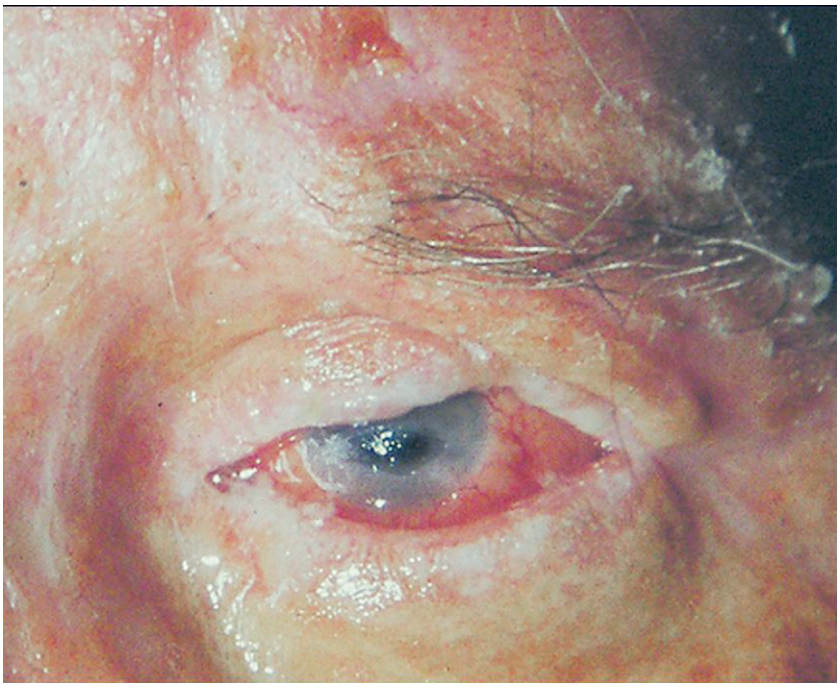

Figure 1. Cicatricial lid changes, lagophthalmos, and corneal surface changes are evident due to the sequelae of herpes zoster ophthalmicus infection. Reproduced with permission from Albert D, Jakobiec F, eds. Principles and Practice of Ophthalmology. Vol. 2. 2nd ed. Philadelphia: Saunders; 2000:846–93.

burning. Because some patients experience hypesthesia of the cornea after HZO, patients may have no ocular discomfort of any kind. The slit-lamp examination may demonstrate an inferior corneal, interpalpebral, or diffuse punctate epithelial keratopathy.

Treatment of mild lagophthalmos or exposure of the cornea may be accomplished with nothing more than ocular surface lubrication, such as artificial teardrops or ophthalmic ointment. When ocular lubrication alone is insufficient to treat the corneal signs and symptoms of lagophthalmos, a surgical treatment that targets the anatomic abnormality causing the lagophthalmos should be planned.

A thorough examination of the eye, lids, and adnexa should be performed. Corneal sensation must be assessed.

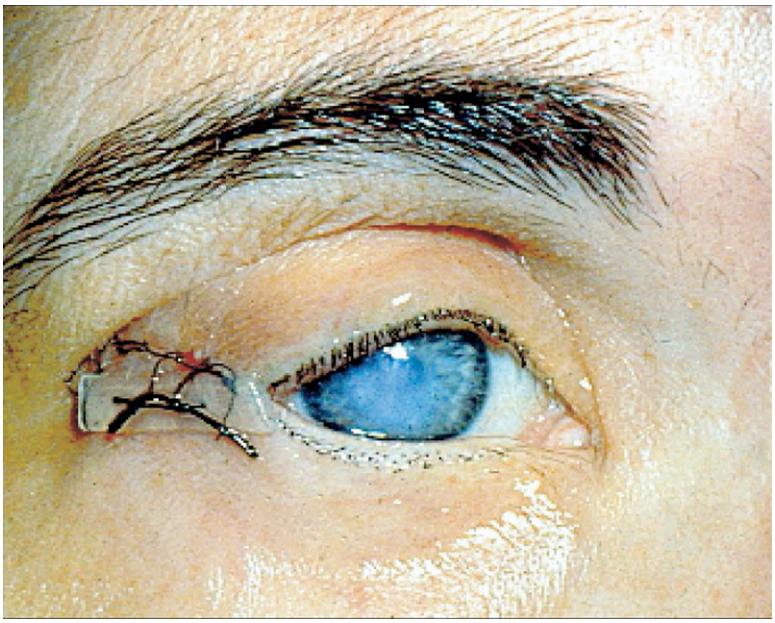

Figure 2. A lateral tarsorrhaphy protects the cornea by reducing corneal exposure. Reproduced with permission from Albert D, Jakobiec F, eds. Principles and Practice of Ophthalmology. Vol. 2. 2nd ed. Philadelphia: Saunders; 2000:846–93.

The degree of lagophthalmos should be documented. Next, orbicularis oculi weakness should be evaluated and differentiated from vertical shortening of the upper eyelid from cicatricial changes. A gross external examination of the lids should be performed by asking the patient to close his or her eyelids gently. Care should be taken to avoid squeezing of the eyelids by the patient, because forceful closure of the eyelids may mask more subtle forms of lagophthalmos.

Orbicularis oculi palsy manifests itself as a weak or incomplete closure of the eyelids. Palsies can be managed in a stepwise fashion, depending on the time course and severity of the problem. Ocular surface lubrication is a first-line treatment for HZO sequelae that is successful in over 50% of cases.[6] Commercially available moisture chambers may also be used to augment the ocular surface lubrication. When clinical signs and symptoms worsen or are not well controlled with ocular lubrication alone, surgical options must be considered. In some cases, orbicularis oculi muscle palsy can be corrected using mechanical means. Both surgically implanted springs and gold weights have been used to achieve eyelid closure. Currently, a gold weight is the most commonly used device to aid eyelid closure. These weights are available in different sizes. The effectiveness of treating the patient's lagophthalmos with a gold weight may be approximated by taping a gold weight to the patient's upper eyelid.[7] Although complications are rare, they include migration of the weight, extrusion, and infection. Townsend reported a success rate of >90% when treating paralytic lagophthalmos with placement of a gold weight.[8] Elevation of the palsied lower lid may also be necessary to achieve an adequate treatment of the lagophthalmos.[9]

If neither ocular lubrication nor placement of a gold weight fully corrects the lagophthalmos, a tarsorrhaphy may become necessary. Depending on the anticipated length of the disability, a temporary or permanent tarsorrhaphy should be performed. A temporary tarsorrhaphy may be used for up to 1 month if the patient's lagophthalmos is expected to be temporary. Temporary tarsorrhaphies are easily performed by placing a horizontal mattress suture with rubber or plastic bolsters through the upper and lower eyelids. The knot should be tied so that the suture can be loosened, the eye examined, and the knot retied if needed (Fig 2). Depending on the degree and location of the keratopathy, a medial, lateral, or central tarsorrhaphy may be performed. Eyedrops may be administered by placing a drop at the medial canthal location and allowing it to seep onto the ocular surface. Other techniques have been developed to replace the use of sutures in temporary tarsorrhaphies. Reinforced tape strips and cyanoacrylate glue have been used to form a temporary tarsorrhaphy. The primary disadvantage of these suture alternatives is that the duration of their effect cannot be accurately predicted. An additional method used to protect the ocular surface is botulinum A toxin, which has been injected into the upper eyelid to produce intentionally a temporary ptosis. This newer technique may provide adequate protection of the ocular surface without the need for suture placement, while still permitting a more predictable duration of the eyelid closure.[10]

Permanent tarsorrhaphy may be necessary when the deficit is expected to persist for more than 1 month, if the patient is unable to use the topical ocular lubricants as needed, or if the temporary tarsorrhaphy cannot remain in place. Like the temporary tarsorrhaphy, a permanent tarsorrhaphy may be placed laterally, medially, laterally and medially, or centrally. The degree of lid closure needed to protect the ocular surface determines how extensive the permanent tarsorrhaphy must be. The goal of this procedure is to produce a permanent firm adhesion between the upper and lower eyelids. This is accomplished by removing the epithelium of the eyelid margins, splitting the anterior and posterior lamellae, and then completing a layered closure of each lamella separately. Permanent tarsorrhaphies can be surgically reversed, but the eyelid margin may sustain damage in the process.

The pillar tarsorrhaphy is an alternative type of permanent tarsorrhaphy that can be beneficial to some patients because it permits an obstructed central cornea with peripheral protection of the ocular surface. Tanenbaum et al described the use of the pillar tarsorrhaphy to treat corneal exposure-related keratopathy of various etiologies. They demonstrated clinical improvement in all 35 cases. This procedure involves the construction of 2 upper eyelid tarsal pillars, which are secured to corresponding locations on the conjunctival side of the lower lid. They demonstrated a narrowed interpalpebral fissure that permitted the reversal of the procedure without lid margin damage. Rare complications associated with this procedure included pyogenic granuloma, stretching of the tarsal pillars, lower eyelid ectropion, and dehiscence of the tarsal pillars.[11]

Complications secondary to severe cicatricial changes, associated with HZO, result from vertical shortening of the eyelids either locally or across the entire width of the eyelid. This type of malformation has been repaired with success by excision of the scars, release of the eyelid traction, and placement of full-thickness skin grafts.[12,13] If a tarsorrhaphy is necessary and the lids are under tension, extra sutures with bolsters may be necessary to achieve lid closure.

Other complications of HZ infection of the lids may be related to the associated necrosis, ischemic vasculitis, or cicatricial changes. These include eyelid malformations such as entropion or ectropion, trichiasis, obliteration of the puncta, and loss of eyelid tissue, which may be repaired by standard oculoplastic procedures.

Conjunctiva

Conjunctivitis from HZO infection can produce a pseudomembranous, membranous, or follicular response. A mucopurulent discharge is commonly evident during the active phase of this disease. Vesicles may be present on the bulbar or palpebral conjunctival surfaces. Normal eye movements may rupture these vesicles (Fig 3). Such a development may result in only a mild inflammatory reaction; however, infection, ulceration, severe scarring, and symblepharon formation may occur in rare instances.[14] Topical antibiotic drops may be administered prophylactically to prevent a secondary bacterial infection, and topical steroids may be used in cases that demonstrate significant inflammation. However, serious conjunctival complications are very rare.

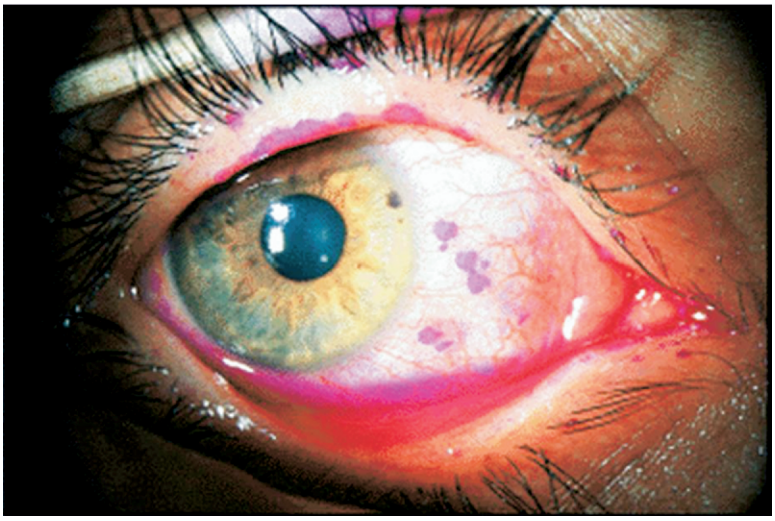

Figure 3. Ruptured conjunctival vesicles are visible with rose bengal stain. Reproduced with permission from Albert D, Jakobiec F, eds. Principles and Practice of Ophthalmology. Vol. 2. 2nd ed. Philadelphia: Saunders; 2000:846–93.

Cornea

The cornea is involved in approximately 65% of patients who develop HZO. A study by Liesegang investigated the incidence of the more common characteristics of zoster-related corneal findings in 94 patients. Of these, he found punctate epithelial keratitis in 51% of patients, early pseudodendrites in 51%, anterior stromal infiltrates in 41%, late corneal mucous plaques in 13%, disciform keratitis in 10%, neurotrophic keratitis in 25%, and exposure keratitis in 11%.[15] His findings supported the common belief that the early lesions were likely due to direct damage from the viral infection, whereas the later sequelae were the result of vasculitis, immune reactions to the viral antigens, delayed hypersensitivity reactions, or damage to the nerves and tissues—although there are some conflicting studies, which will be discussed below.

Thus, corneal damage from HZO may occur in the early stages of the infection, or months or even years later. Despite appropriate medical and surgical management, significant ocular damage and loss of vision may result.

Mucous Plaque and Stromal Keratitis

Marsh[16] and Piebanga and Laibson[17] described some of the late corneal disease associated with HZ, including a chronic epithelial keratitis that was characterized by epithelial mucous plaques, which have also been termed mucous plaque keratitis (MPK). These lesions typically appear months after the initial presentation of the corneal disease. The plaques varied in size and shape and changed from day to day. Mucous plaques can be debrided without damaging the underlying epithelium.

Marsh and Cooper noted that MPK generally developed within 2 years of the initial VZV infection of the eye and is commonly associated with a peripheral keratitis, stromal keratitis, decreased corneal sensation, anterior segment inflammation, increased intraocular pressure (IOP), and cataracts. They studied this condition in 1221 patients and noted that MPK responded well to topical steroid drops and acetylcysteine 10% drops; however, surgical intervention was sometimes necessary to treat associated neurotrophic keratitis, glaucoma, or cataracts.[18] Cobo noted that the treatment of MPK sometimes required chronic topical steroid treatment.[19] It is important to inform the patient concerning the risks associated with corticosteroid use, which include cataract formation and increased IOP. Patients on topical steroids should be evaluated frequently to assess their IOP and the status of the inflammatory condition. Additionally, the risk of corneal melting is significant in this group because of corneal exposure and the hypesthesia that is common in these patients. Although steroids are sometimes necessary, the risk of keratolysis is always present, even when low-potency steroids are used infrequently.[20]

Past studies failed to demonstrate live virus by culture in cases of MPK.[15,17,21] The role of general antiviral treatment of MPK is not settled, but there is significant evidence that these lesions may contain live virus. Pavan-Langston et al performed polymerase chain reaction studies on 1 patient's excised corneal button and the response of 4 patients to antiviral therapy. They concluded that recurrent viral infection by VZV may play a role in this late manifestation of the disease, associated with MPK; thus, specific antiviral therapy may be warranted in the treatment of MPK. Specific antiviral therapy was not consistently successful in all cases, but topical trifluridine, vidarabine, and oral acyclovir were individually used successfully in different cases.[22] The use of newer topical and oral antivirals may improve the general success rate of treatment.

Late Keratitis

Late-stage stromal keratitis can occur months after the initial onset of the zoster corneal infection. This condition is characterized by deep stromal inflammation, which presents

as a typical disciform lesion or as a peripheral keratitis. Corneal edema may be associated with these lesions. If these lesions are not treated with corticosteroids, a chronic inflammation may develop that leads to neovascularization, scarring, and ulceration of the cornea. Later, lipid deposition may occur. Corticosteroids are used to treat the underlying inflammatory cause of these disorders.

Additionally, Reijo et al described endotheliitis and subsequent endothelial cell loss associated with late stromal keratitis and keratouveitis.[23] If significant endothelial cell loss develops, a penetrating keratoplasty (PK) or Descemet's stripping and automated endothelial keratoplasty procedure may be necessary. Corneal exposure, neurotrophic keratitis, and other conditions, which may compromise the new corneal graft, must be addressed before corneal transplantation.

Five of the 10 Reijo et al study patients developed concurrent increased IOP.[23] Increased IOP associated with anterior segment inflammation from zoster can be difficult to treat and long lasting. Topical β-blockers, α-agonist medications, and carbonic anhydrase inhibitors can be used to control IOP, but surgery may become necessary in some cases. Prostaglandin analogs are usually avoided as they may increase the intraocular inflammation.

Neurotrophic Keratitis

Because VZV infections affect sensory nerves, some patients experience a certain degree of hypesthesia. The degree of sensory loss in the cornea varies, and the length of time that the deficit persists is unpredictable. Long-term observation is warranted, because significant improvement can occur over the course of months. Cobo et al determined that half of all patients with HZO experience some degree of corneal hypesthesia. They also found that the mean time of presentation of the corneal hypesthesia was 3 days after onset of the VZV infection.[24] Unfortunately, it is not possible to predict the degree or course of corneal sensory recovery.

A lack of corneal sensation reduces blinking and results in a secondary corneal exposure and dry eye. Dry eyes and their associated pathology may go undiagnosed in the HZO patient. These patients' eyes should be scrutinized for signs of corneal epithelial breakdown, exposure, and lagophthalmos. These conditions cause breakdown of the corneal epithelium, with secondary inflammation, thinning, and possible secondary infections. As the cornea dries, the surface becomes irregular, which would normally cause decreased visual acuity (VA), but because of the corneal pseudodendrites, keratitis, inflammation, and associated loss of stromal clarity, the patient may not notice any change in VA. Furthermore, these patients have little or no discomfort because of their hypesthesia. These factors frequently result in progression of the neurotrophic keratitis without any realization of the worsening condition by the patient.

As the corneal epithelium deteriorates, a punctate keratopathy can be seen. If the epithelial breakdown progresses, large corneal epithelial defects occur. Corneal ulcerations occur that are sterile in nature but can become infected. If untreated, the cornea may become opaque, continue to thin, and eventually perforate (Fig 4). This condition is treated similarly to lagophthalmos; however, close observation is necessary to provide the appropriate treatment for the keratopathy.

Chen et al studied the use of amniotic membrane (AM) grafts for the treatment of severe neurotrophic corneal ulcers in 15 patients who developed a neurotrophic cornea after HZO, PK for corneal scars or ulcers due to previous neurotrophic keratitis, diabetes mellitus, radiation, and neurosurgical nerve damage. After a mean follow-up time of 18 months, 12 eyes achieved rapid reepithelialization and healed with reduced inflammation. Of the 4 remaining eyes,

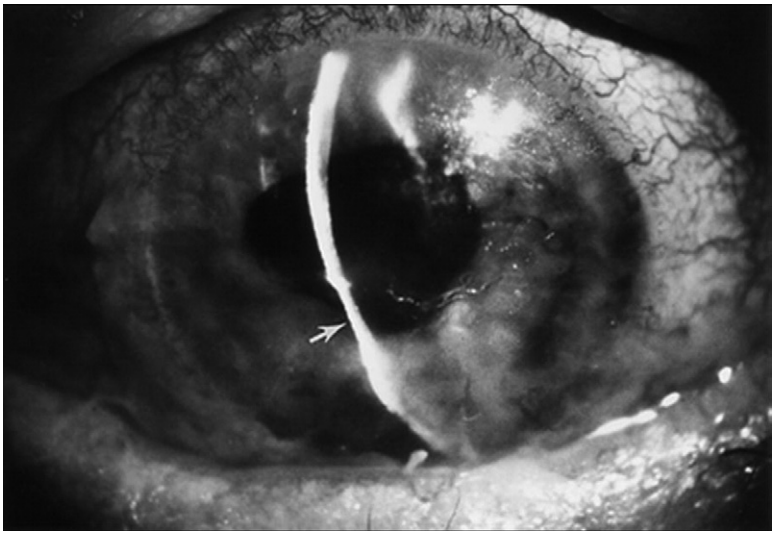

Figure 4. Neurotrophic keratitis can result in corneal ulcerations and thinning, which are sterile in nature. An irregular corneal surface is shown, with corneal thinning (arrow). Reproduced with permission from Albert D, Jakobiec F, eds. Principles and Practice of Ophthalmology. Vol. 2. 2nd ed. Philadelphia: Saunders; 2000:846–93.

3 required a tarsorrhaphy and 1 required a PK and tarsorrhaphy. All corneal surfaces healed at the termination of the study, with 8 eyes demonstrating improved VA. Although further studies are needed, this technique may provide a surgical alternative and be used as adjunct therapy with other procedures.[25] Additionally, autologous serum eyedrops may provide an added benefit to the ocular surface and aid in healing of the corneal epithelium, but more studies are needed.[26–28]

Corneal Perforations

Small corneal perforations of approximately 1.5 mm can be treated emergently by using cyanoacrylate glue or fibrin-based tissue adhesives.[29–31] The use of these materials is off label. Careful observation is required because the duration of the glue is unpredictable. Additionally, use of a medication to lower IOP may facilitate closure of the corneal perforation. The purpose of using these materials is to allow the cornea to heal the small corneal defect. If the defect is larger than what can be safely treated with a tissue adhesive, a patch graft to the cornea is needed. Interrupted sutures should be used to avoid total loss of the graft's sutures if one single region thins and the sutures become loose, break, or cheesewire through the tissue. Single-layer and multilayer AM grafts have been successfully used to close corneal perforations as large as 1.5 to 2.0 mm. These materials have been used with fibrin tissue adhesive, to seal the defect, and with sutured AM alone.[32–34] Investigators demonstrated an increased rate of corneal reepithelialization and reduced rate of corneal melts with AM transplants.[35] As in the use of AM transplants in the treatment of neurotrophic corneal ulcers, AMs have been shown to decrease metalloproteinase activity and increase tissue inhibitors of metalloproteinase.[36] Thus, AM transplants may provide not only a physical tissue plug, but also a scaffolding for reepithelialization and an ability to reduce further corneal tissue damage by decreasing metalloproteinase activity.

To create an environment that will promote healing, the underlying conditions responsible for the perforation must be corrected. Dry eyes frequently coexist with other sequelae of HZO. If the dry eye is only temporary, it may be possible to use copious ocular lubricants, such as preservative-free ointments and viscous artificial teardrops, until the eye heals and corneal sensation returns. Punctal plugs may be used with ocular lubricants as an adjunct therapy or may, on rare occasions, provide sufficient therapy alone. Although tears and punctal plugs would seem to be preferable to surgery, a partial or complete tarsorrhaphy may provide the best chance for corneal healing. As was discussed earlier, lid abnormalities must be corrected before any action is taken, and then a central, medial, lateral, medial and lateral, or complete tarsorrhaphy can be performed. Although tarsorrhaphy is a very basic procedure, this underutilized surgery can provide protection of the ocular surface until the cornea heals, corneal sensation returns, or another corneal procedure, such as a keratoprosthesis placement, can be performed.

If significant corneal scarring exists in the presence of a dry eye, a total conjunctival flap can also be used to provide corneal surface protection and to help heal the ocular surface. The conjunctival graft is pulled over the cornea and sutured over the inferior limbus with interrupted sutures. Care must be taken to avoid a buttonhole in the graft, because this will typically expand and render the graft ineffective. If corneal sensation returns, the graft can be removed or a PK can be performed through the total conjunctival graft.

A previously unreported technique may also be helpful to close a corneal defect and protect the cornea. This involves the construction of a pedicle graft from the upper lid. This type of lid-sharing technique for the cornea uses a piece of upper eyelid conjunctiva and small piece of tarsus to form a pedicle, which is sewn over the corneal defect. The pedicle is left attached to the upper lid. This procedure provides a vascularized conjunctival graft and partially covers the affected area with the lid. If corneal sensation returns and the cornea heals, the graft can be reversed (Kaufman HE, personal communication, December 2005).

Corneal grafts for restoration of corneal clarity are discussed below.

Peripheral Corneal Ulcers

Peripheral corneal ulcers are a rare complication associated with VZV corneal infections. The ulcers may resemble Mooren ulcers and are typically associated with anterior uveitis or stromal keratitis, but regional exposure and neurotrophic keratitis must be considered. These perilimbal lesions may be controlled by conventional measures, but severe cases have been reported that resulted in loss of the entire cornea.[37] Treatment typically involves control of any anterior segment inflammation or stromal keratitis. Such control can usually be achieved with the use of topical steroids. If the peripheral corneal ulcers continue to progress: first, consider keratolysis from topical steroid use and reduce or eliminate steroids; next, consider a conjunctival graft, which may stabilize the cornea and help to halt the progression of the corneal thinning.

Alino et al reviewed the use of conjunctival grafts for HZ complications and other corneal disorders. The majority of the partial conjunctival flaps were successful, but 2 of 13 patients required repeated grafts due to retraction of the flaps. One of their patients, whose graft retracted, suffered a corneal perforation requiring a PK, and the other patient, whose graft retracted, suffered recurrent ulcerations and required 2 subsequent conjunctival regrafts.[38] Their retrospective study supported the use of conjunctival grafts for persistent nonhealing epithelial defects.

To accomplish a successful graft, the surface of the ulcer must be cleaned of any necrotic debris and the corneal epithelium removed from the region of the planned conjunctival graft site. If the epithelium is left in place, the conjunctival graft will not adhere to the surface of the cornea. Next, the adjacent conjunctiva can be mobilized by performing a localized peritomy and sliding the conjunctiva over the affected area. The conjunctiva is then sutured to the cornea with 10-0 interrupted nylon sutures. Interrupted sutures are preferred so that the conjunctival graft will stay in

place if the sutures loosen or are lost in one area. Use of an AM may provide protection to the conjunctival graft and may reduce localized inflammation. Additionally, it may also be possible to use a fibrin-based tissue adhesive to anchor these conjunctival and AM grafts, but these measures have not been extensively studied.

If the cause of the peripheral corneal ulcers is secondary to exposure or neurotrophic keratitis, a tarsorrhaphy may be necessary, with or without a local peripheral conjunctival graft.

Corneal Scars

Because the cornea is commonly involved in cases of HZO, corneal scarring after a VZV corneal infection is common. The corneal scar may manifest as a faint stromal haze or may be seen as an opaque region of the cornea associated with corneal thinning. Careful assessment of the scar is necessary and should include measuring total corneal thickness centrally and in the region of the scar. The depth of the scar should then be measured. This may be accomplished with an optical pachymeter, confocal microscope, or ultrasonic biomicroscope. If the residual corneal thickness, posterior to the scar, is <250 μm and the corneal endothelium is compromised, a PK will be necessary. If, however, the corneal endothelium is normal, then a PK or deep lamellar anterior keratoplasty can be performed to restore corneal clarity. If the corneal scar is dense, the condition of the endothelium may be difficult to assess because the scar may mask corneal edema and direct visualization of the corneal endothelium may not be possible.

It is important to remember that the risk of corneal ectasia after lamellar keratectomy increases when the residual corneal bed (thickness of the cornea posterior to the scar or lamellar keratectomy) is <250 μm. Corneal sutures may help to increase fibrosis after a corneal graft and thus reduce the risk of ectasia, but this has not been established.

Corneal scars in the anterior half of the cornea can be treated by keratectomy or by lamellar keratoplasty. The keratectomy may be performed using a microkeratome or excimer laser phototherapeutic keratectomy (PTK). Freehand keratectomy can be performed in the periphery of the cornea; however, the automated methods provide a relatively smoother surface to the bed.

Phototherapeutic keratectomy should not be used in the face of an active infection, but it has been used to treat postinfection scars. Many studies concerning the treatment of postherpetic scars by PTK have been published, with conflicting results. Four prominent studies noted a range of improved VA in 25% to 100% of patients.[25,39–41] The wide disparity in the success rates of these studies reflects the controversy associated with this topic. These findings may be explained by a different ablation rate with the scar tissue compared with the adjacent tissue or the inability to perform accurate wavefront analysis through a corneal scar.

Corneal scarring has been reported after manual keratectomy and PTK; however, topical steroids and the judicious use of mitomycin C 0.02% for 12 seconds can be used to reduce the risk of corneal scarring after this surgery.[42] The mitomycin C may be applied via a round 6-mm pupil occluder sponge after removal of the corneal scar. The mitomycin C–soaked sponge is removed, and the eye is then thoroughly flushed with ophthalmic balanced salt solution to remove the mitomycin C. A bandage contact lens is applied for comfort. The typical postoperative medication regimen involves a topical steroid, antibiotic, and nonviscous ophthalmic formulation of a nonsteroidal antiinflammatory drug, used 4 times a day for a week until the surface is epithelialized. The steroid may be tapered over 3 to 4 months.

One important caveat must be emphasized: Any preexisting ocular inflammation, increased IOP, dry eye, exposure, or neurotrophic keratitis must be treated and under good control before attempting any corneal grafts to restore the clarity of the cornea. If these conditions are not addressed and controlled, the risk of failure is very high.

Role of Keratoprostheses

A keratoprosthesis may be the final option for patients whose ocular inflammation is well controlled but who suffer dry eyes and multiple graft rejections. Although the Alphacor type of keratoprosthesis is not generally successful in patients with dry eyes, the Boston keratoprosthesis, type II, and Cardona through-the-lid type of keratoprosthesis provide the subject with a possible treatment option.[43] These devices can impart good vision but are not considered a first-line treatment and introduce their own unique set of complications.

Additional Ophthalmic Surgery after Herpes Zoster Ophthalmicus

Because of the multiple complications associated with VZV infection of the eye, other ocular surgery may be necessary at some point in the zoster patient. If the patient develops increased IOP and anterior segment inflammation, cataracts are common, as is trabeculectomy. The use of steroids in HZO is frequently unavoidable; therefore, cataract extraction may be necessary. Also, various types of corneal grafts may be used to restore corneal clarity.

If coexisting lid, IOP, tear film, and corneal sensation deficits are addressed properly and ocular inflammation is controlled, then other types of ocular surgeries have a high success rate.[22] Marsh and Cooper found that the surgical results for corneal scars, glaucoma, and cataracts were no different between HZO patients and routine cases, except that they noted a tendency for prolonged inflammation after surgery in HZO patients. Additionally, they did not hesitate to place a tarsorrhaphy if healing was delayed.[44] Like Marsh and Cooper, other groups report good success rates with corneal surgery in patients who have had HZO. A common thread that runs through these studies is the importance of partial tarsorrhaphies when needed, frequent ocular surface lubrication, and careful postoperative management.[45,46] Additionally, preoperative treatment with a topical antiinflammatory may reduce the degree of postoperative inflammation and improve the success rate of post-HZO ocular surgeries.

Conclusion

Herpes zoster ophthalmicus commonly causes complications that can result in significant ocular and visual morbidity. The postinfection complications affect all regions of the eye and adnexa, including the cornea, conjunctiva, and lids. Despite appropriate and timely treatment, ocular damage and loss of vision may still result. A higher rate of treatment successes for VZV-related complications was obtained when any preexisting ocular inflammation, increased IOP, lagophthalmos, dry eye, exposure, or neurotrophic keratitis was treated and under control before attempting ocular surgery. If these conditions are not addressed and controlled, the risk of failure is very high.

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