

InTown Veterinary Group Newsletter

Volume 8, Issue 4
 October 2008

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Upcoming Doctor Continuing Education Lectures:

For an always current list of upcoming CE, go to www.InTownVet.com & click on CE Lectures under "veterinarians".

Upcoming Lectures:

Tues. Oct. 28 - Common Surgical Diseases of the Upper Respiratory Tract in Dogs and Cats, at Port City, Portsmouth, NH.

Wed. Nov. 12 - Diagnosing and Treating Congestive Heart Failure in Dogs and Cats: New Options, at Mass Vet, Woburn, MA.

Tues. Dec. 9 - Lactate Physiology and Clinical Applications, at Mass Vet in Woburn, MA.



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■ Port City Veterinary Referral Hospital
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Laryngeal Paralysis



Karen F. Pastor, DVM, DACVS

The high heat and humidity of summer weather is gone but veterinarians and owners must continue to be on the lookout for cases of laryngeal paralysis (LP). There is no evidence of a seasonal primary reason for laryngeal paralysis. This article reviews the pathophysiology, diagnosis, and treatment of laryngeal paralysis in dogs and cats. LP is a common disease in older large and giant breed dogs. In dogs, the diagnosis is straightforward. In cats, the disease is rare and can be challenging. In both dogs and cats, the treatment of choice is unilateral arytenoid lateralization (“tie back” surgery).

The cricoarytenoideus dorsalis muscle is the only abductor muscle of the rima glottidis and is innervated by the recurrent laryngeal nerve. The recurrent laryngeal nerve is a branch of the vagus nerve, which runs dorsolateral to the trachea and terminates as the caudal laryngeal nerve. It innervates all the muscles of the larynx except for the cricothyroid muscle.

The larynx has three functions:

- **Respiration:** During inspiration, the arytenoids abduct and increase the size of the rima glottidis (narrowest part of the laryngeal airway). It is normally elongated and diamond shaped. During expiration, the arytenoids passively return to the resting position. During exercise, the arytenoid abduction is sustained during both inspiration and expiration to maximize airflow and minimize airway resistance.
- **Deglutition:** During swallowing, the reflex closure of the larynx prevents aspiration of food and fluid to the airway.
- **Vocalization:** Voice production is related to the movement of air over the vocal and vestibular folds, and their change in length and thickness produced by the contraction of the laryngeal muscles.

Laryngeal paralysis and dysfunction is the cause of the following clinical signs. Respiratory dysfunction occurs due to decreased resting rima glottidis size and leads to increased airflow resistance and turbulence, resulting in an audible laryngeal stridor. Loss of laryngeal abduction on inspiration and loss of sustained laryngeal abduction with exercise leads to upper airway obstruction worsening with exercise. Upper airway obstruction leads to laryngeal edema and dynamic laryngeal collapse. Swallowing dysfunction occurs due to loss of laryngeal adduction, reduced airway protection and leads to aspiration pneumonia. Altered vocalization also occurs in most cases. In dogs the bark may become hoarse; in cats, there will be a change in the sound of the meow, or an absence of purring.

The mechanism of idiopathic laryngeal paralysis in dogs is described as a progressive, noninflammatory degenerative disease of the recurrent laryngeal nerve. Histopathologic characteristics of the recurrent laryngeal nerve include loss of axons, beading of myelin, and perineural fibrosis. Neurogenic atrophy of the cricoarytenoideus dorsalis muscle has been noted in studies (Griffen JF, Krahwinkel DJ).

Laryngeal paralysis can be unilateral or bilateral, congenital or acquired. Congenital LP occurs in the Siberian Husky, Bouvier des Flandres, Staffordshire Bull Terrier and Dalmatian. Acquired LP often occurs in middle age to older large breed dogs. Males are slightly more affected than females. In cats, there is no breed, age or sex predilection.

Causes of acquired LP in dogs are central or peripheral vagal nerve lesions, recurrent or caudal laryngeal nerve lesions, trauma to neck (dog bites, penetrating wounds, foreign bodies), iatrogenic trauma secondary to previous neck surgery, intrathoracic and extrathoracic masses, neuropathy, myopathy, and idiopathic. Idiopathic is the most common cause of acquired LP. Other rare causes in dogs are hypodrenocorticism, organophosphate poisoning, and CNS, posterior brain lesions. Causes of laryngeal paralysis in cats are polyneuropathy, neoplasia (lymphosarcoma

affecting the vagus and laryngeal nerves), trauma to neck and iatrogenic trauma from previous neck surgery (thyroid, neck tumors, trachea). Fifty percent of LP cases in cats are idiopathic.

Clinical signs in dogs include laryngeal stridor, exercise intolerance, gagging, coughing, dysphagia, vomiting, hoarse bark or voice change, dyspnea, cyanosis and syncope.

Clinical signs in cats include laryngeal stridor, tachypnea, dyspnea, dysphagia, weight loss, coughing, absence of purring and change in meow.

Definitive diagnosis is by direct laryngoscopy. Laryngoscopy is performed with the animal under a light plane of anesthesia. The potential problem with diagnosing laryngeal paralysis is that anesthetic agents normally depress laryngeal movement. Butorphanol (0.2 mg/kg IM) can be give with propofol IV (given slowly to effect). In addition, dopram (1 mg/kg IV) can be given to enhance inspiratory and expiratory movements. The laryngoscopy exam demonstrates the inability of the arytenoid cartilage to abduct and open the glottidis during inspiration (figures 1-3). There may be fluttering of the arytenoids and/or vocal folds. The larynx may be edematous and erythematous. A definitive diagnosis in cats can be more difficult due to typical feline laryngospasm.

Transnasal laryngoscopy in dogs has been recently described as a successful diagnostic tool. It is an

adaptation of the well-accepted means of diagnosing LP in horses. A 2.5mm flexible endoscope is passed through the nasal passages of dogs treated with intranasal lidocaine. Limitations of this technique include the need for a small, flexible endoscope and a large patient.

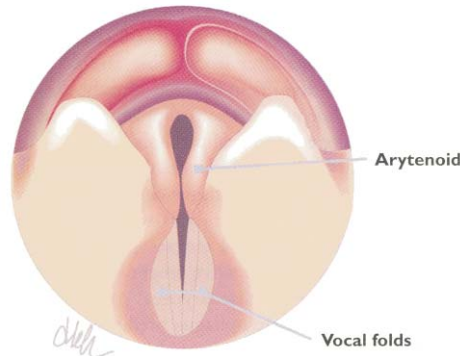


Fig 1: Drawing of the normal Larynx as seen via laryngoscopy.

Source: *Compend Contin. Educ Pract Vet*, Vol 6, No. 4:2005, p860.

Minimum database for an animal in which you suspect laryngeal paralysis includes a physical exam, neurologic exam, CBC, chemistry profile, urinalysis, thyroid profile and thoracic radiographs (ventral/dorsal, lateral, and lateral cervical neck views) to rule out non-idiopathic causes of laryngeal paralysis.

Good quality radiographic technique and thorough assessment is critical to rule out other causes of LP. In one study, 70% of dogs confirmed with LP had abnormal thoracic radiographs (20% had megaesophagus and 15% had aspiration pneumonia). Other abnormalities to look for are intrathoracic and extrathoracic masses, pulmonary edema, and pulmonary metastases.

The normal larynx is air filled. Loss of the normal air density in the larynx may indicate a laryngeal mass or edema. The association between LP and hypothyroidism is unclear. Treating the hypothyroidism will not reverse the LP. Additional tests (although not necessary) are EKG and echocardiogram to rule out heart disease, thoracic ultrasound to evaluate for masses, EMG to aid with diffuse myopathies, histopathology of affected muscles to confirm denervation atrophy (biopsy of intrinsic laryngeal muscles), and endocrinologic screening.

The treatment of laryngeal paralysis on an emergency basis is sedation, cage rest, and oxygen. Morphine (.05-.1mg/kg IM) is the preferred sedative as it alleviates the anxiety and air hunger of acute upper airway obstruction. Morphine can be combined with acepromazine (.01-.02mg/kg IM) to reduce anxiety. In addition, many of these animals need to be treated symptomatically for hyperthermia and heatstroke resulting from the inability to adequately ventilate through panting. Occasionally, general anesthesia (propofol IV), endotracheal intubation and temporary tracheostomy are required to stabilize these animals.

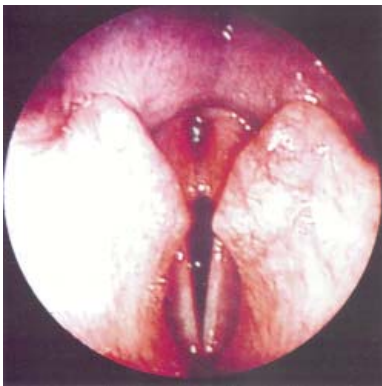


Fig 2: Laryngoscopic view of a patient with bilateral laryngeal paralysis.

Source: *Compend Contin. Educ Pract Vet*, Vol 6, No. 4:2005, p860.

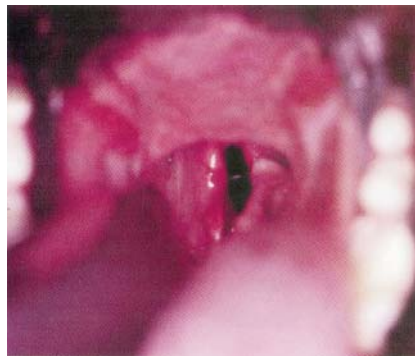


Fig 3: Laryngoscopic view of a patient with unilateral laryngeal paralysis.

Source: *Compend Contin. Educ Pract Vet*, Vol 6, No. 4:2005, p861.

Medical management may be enough for mild cases of laryngeal paralysis. Medical management includes weight loss, exercise restriction, the avoidance of stress and excitement, and a cool environment.

Surgical treatment of choice for laryngeal paralysis is unilateral arytenoid lateralization. The goal is to enlarge the glottidis without exaggerating aspiration of food and saliva. A permanent suture is placed between the arytenoid and cricoid cartilage, which enlarges the airway (figure 4).

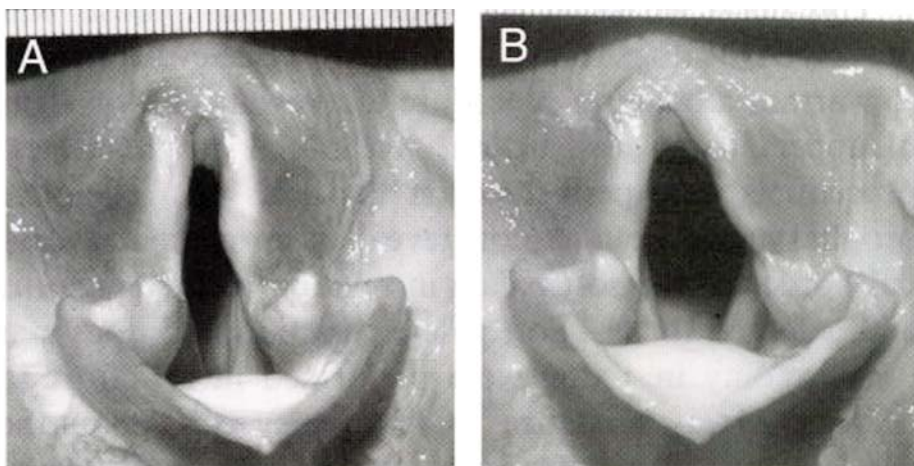


Fig 4: Cadaver Specimen. Before (fig A) and After (fig B) unilateral cricoarytenoid lateralization.

Source: *Veterinary Surgery* 36: p523, 2007

A study comparing the cricoarytenoid and thyroarytenoid lateralization techniques was performed. Findings indicated that the cricoarytenoid lateralization provided a greater increase in the size of the glottidis opening (207%), versus the thyroarytenoid lateralization. The two major advantages of the unilateral arytenoid lateralization are:

1. A temporary tracheostomy is not needed.
2. Laryngeal webbing does not occur because the laryngeal mucosa is not invaded.

Bilateral arytenoid lateralization can be carried out, but it is unnecessary and carries a much higher complication rate. Partial laryngectomy is another surgical option but it is not recommended at this time due to the high incidence of postoperative complications including laryngeal scar tissue (laryngeal webbing). A permanent tracheostomy is not recommended for laryngeal paralysis. It is however recommended for laryngeal collapse, which occurs most commonly in brachiocephalic breeds.

Brachiocephalic breeds have chronic laryngeal changes secondary to elongated soft palate, stenotic nares,

everted saccules, and hypoplastic trachea. These chronic changes lead to laryngeal collapse. Fortunately, LP is not common in these breeds.

Postoperative care includes close observation of respiratory distress, steroids to alleviate laryngeal edema, NPO for 12 hours then elevate food and water and monitor for signs of aspiration pneumonia. Most animals are discharged within 24 - 48 hours postoperative.

The most common postoperative complication is aspiration pneumonia. Aspiration pneumonia has been reported in 18% of cases. The majority of these cases recover with treatment. In addition, there may be recurrence of clinical signs due to suture breakage or pullout secondary to cartilage fracture. Other complications include gagging, coughing, infection and seroma.

Long-term care of these animals is necessary. Long term care consists of no neck leads or collars, use of a harness, always offering food and water in an elevated position and minimal exercise on hot, humid days. For the majority of animals treated with unilateral arytenoid lateralization for laryngeal paralysis, the outcome is good. Owners claim that their dogs are significantly improved and are "new dogs". Median survival in one large retrospective study was > 1,000 days with most animals dying from unrelated causes. ■

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Corneal Ulcers: the Simple, the Scary, and the Stubborn

Nancy B. Cottrill, DVM, MS, DACVO

It is not the “*what*” to use to treat a corneal ulcer that is the crux of the problem. Rather, it is the “*why*”, as in: “Why does this pet have a corneal ulcer?” Once you determine the cause of the ulcer, an effective treatment plan can follow and future ulcers can be prevented. You can be a corneal superhero to your clients and patients!

Helpful rules of thumb rules of thumb include:

1. Always look for the cause of the corneal ulcer using a bright light and magnification.
2. If the ulcer does not heal within one week, look again.
3. If you cannot identify a cause and the ulcer has not healed in one week, call an ophthalmologist or offer a referral. Valuable time, vision and even an eye can be lost if there is a delay.

Diagnostic Aids

Helpful diagnostic aids include:

- Fluorescein stain
- Rose Bengal stain
- magnification (loupe, ophthalmoscope)
- a light source
- a darkened room

Fluorescein stain is hydrophilic and will detect lesions that extend through all 5-7 layers of the epithelium. Rose Bengal stain is a vital stain that detects punctate and dendritic erosions of Feline Herpes Virus (FHV). These erosions only involve the epithelium and do not breach the stroma initially.

Corneal Healing

Corneal healing occurs in a three-step process:

1. Migration of cells to cover the defect,
2. Mitosis of cells to complete the healing,
3. Maturation of the cells so that they can resist infections and are securely bonded.

When corneas go bad and the stroma (or worse) becomes involved, bacterial invasion can occur, followed by tissue destruction, and even corneal perforation. An uncomplicated corneal ulcer will heal at the rate of 1 mm/day.

Causes of Corneal Ulcers

Think “outward in” starting with the eyelids. Adnexal abnormalities include entropion, distichia, trichiasis, lid masses, ectopic cilia and chalazia with ectopic cilia. Adnexal abnormalities typically occur in younger patients. Inadequate lid function can lead to ulcers as well, including lagophthalmos and facial nerve paralysis. Tear film problems include KCS and Mucin Tear Film Deficiency. Corneal foreign bodies, chemical abrasion, and trauma are potential causes. Viral causes are common in cats. Bacteria rarely cause corneal ulcers, but can infect a corneal ulcer. Fungal ulcers are very uncommon in small animals. Older dogs, especially patients with Cushing’s syndrome, hypercalcemia, or renal disease may have abnormal mineral deposits in their corneas causing pain and ulceration. Corneal endothelial degeneration is an aging change, or hereditary in some breeds, causing bullae formation that can then rupture to cause an ulcer. Finally, there are the indolent ulcers that do not always have an identifiable cause and do not heal in a normal amount of time.

Adnexal Abnormalities

Entropion is the condition in which the eyelids roll in and irritate and/or ulcerate the cornea. In puppies less than 5-6 months old and in spastic entropion, tacking the eyelids is the best temporary solution.

Distichia are eyelashes that emerge from the lid Meibomian gland openings on the lid margin and may abrade the cornea. Surgical options to treat distichia include cryosurgery, dermal biopsy punch excision of the hair follicle base, and electroepilation.

Trichiasis is the condition where cilia in a normal location curl or are directed toward the cornea. Facial fold trichiasis can cause ulceration, most commonly in Pekingese and Pugs.

Ectopic cilia grow through the inside of the eyelid and abrade the cornea to cause ulcers that may be vertical in orientation. The conjunctival plug containing the hair follicle should be excised and the base treated with cryosurgery.

Chalazia +/- cilia: Chalazia are styes, or blocked Meibomian (oil) glands, that irritate the cornea. These can be warm compressed to help resolve them, but usually need to be incised and curetted for resolution.

Eyelid masses: Eyelid masses should be removed, not "watched"! A four-sided ("house") excision is best. Biopsy and cryosurgery is another option, however there is a 30% rate of recurrence compared to a 5% chance with proper excision.

Lagophthalmos/Macroblepharon:

In many brachycephalic breeds, the eyelids do not close completely, leaving the central cornea to desiccate or ulcerate. A medial a/o lateral canthoplasty to shorten the eyelid opening is the remedy.

Facial Nerve Paralysis: Therapy for this condition includes corneal lubrication, tear stimulation since the tear production is often decreased as well, and a partial temporary, or permanent tarsorrhaphy to help avoid ulceration.

Trauma: Blunt or sharp trauma can cause a corneal laceration. These are best referred for primary closure with 8-0 or 9-0 suture, and perhaps a graft, under an operating microscope.

Corneal foreign body: These may be either embedded in the cornea, or may penetrate into the anterior chamber. These cases are best referred, rather than risk encountering a perforation or inadvertently pushing them deeper.

Tear Film Abnormalities

KCS: KCS is often under-diagnosed. Tear production of <15 mm/min is too low, especially in the face of an ulcer or inflammation. High quality tear supplements, preferably free of preservatives, should be used. Cyclosporine therapy should be instituted in the ulcerated eye, depending on the degree of ulceration and tear deficiency, and in the fellow eye if dry. KCS in cats is often a result of Feline Herpes Virus infection. Topical cyclosporine therapy is usually contraindicated

since it will further suppress the local immune system and promote a herpes outbreak.

Mucin Tear Film Deficiency: This condition mimics the appearance of KCS, however the Schirmer tear test result is within normal range. This is treated with cyclosporine to stimulate the goblet cells to produce mucin.

Other Causes of Corneal Ulcers: Chemicals such as shampoos, cleaners, and ear medications may cause ulcers. Infectious causes in dogs are rare. Bacterial infections do not typically cause ulceration, but may infect a corneal ulcer. Feline Herpes Virus is the most common cause of corneal ulceration in cats and should be treated with topical anti-viral medications.

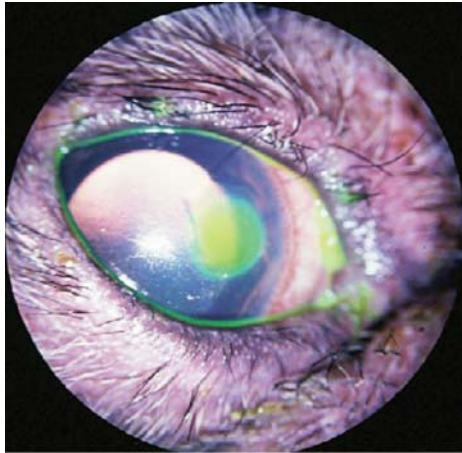


Fig. 1: Superficial Corneal Ulcer

Treatment of superficial corneal ulcers in dogs

After the cause is identified and treated, use topical antibiotic solution QID. Triple antibiotic is a good choice. Atropine solution should be used BID initially, then SID until healed. The cycloplegic-mydriatic effects of atropine block ciliary spasms, pain, dilate the pupil to decrease synechiae formation, and help return the uveal tract vessels to normal permeability. An Elizabethan collar should be worn at all times (24/7). Recheck the eye in 3-4 days.

Anti-viral therapy for FHV ulcers in cats

Topical antiviral therapy is crucial. Several antiviral solutions are available including Cidofovir, Idoxuridine, and Viroptic (triflurthymidine). Cidofovir is used BID x 2 wks at a cost of \$70/5 ml. Cidofovir must be compounded. Idoxuridine is used q 4 h x 48 hrs, then QID x 2 wk at a cost of \$60/15 ml. Idoxuridine may sting and must be compounded. Viroptic (triflurthymidine) is used q 3 h x 48 hrs, then QID x 2 wks. It may sting both the eye and the pocketbook as its cost is \$110/7.5 ml.

Adjunctive therapy for FHV ulcers

Terramycin or Erythromycin ointment are used TID for secondary bacterial infection. The usual suspects are Mycoplasma or Chlamydia. Atropine ointment is used for pain. Ointment travels down the nasolacrimal duct less than solution does resulting in less drooling due to the bitter taste. An Elizabethan collar should be in place at all times. FHV ulcers should be rechecked in 3-4 days if superficial.

Treatment of corneal ulcers in brachycephalic dogs

The scary thing is that even superficial ulcers can progress and perforate in 24 hours! Many brachycephalic dogs have KCS and lid abnormalities. For the first 24 hrs, treat aggressively q 2 h with acetylcysteine/Mucomyst® in tears w/ a final concentration of 5-8%, autologous serum, and ciprofloxacin solution (good against Pseudomonas). Topical atropine solution should be used q 4-12 hrs. depending on the degree of miosis and presence of KCS. An Elizabethan collar should be worn and the ulcer rechecked in 1-2 days.

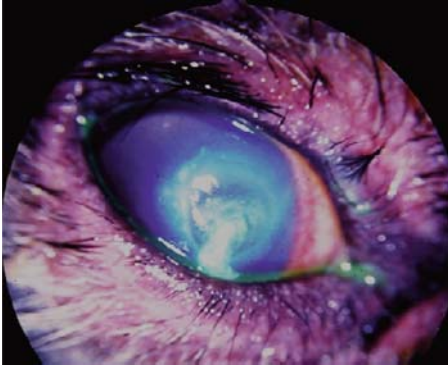


Fig 2: Corneal Perforation

Treatment of deep ulcers, descemetocoeles, and melting ulcers

It is best to refer these cases ASAP and BIP (Before It Perforates)! These cases are hospitalized and treated as for a brachycephalic ulcer. If they are not taken to surgery for a graft (conjunctival, lamellar corneal, or BioSIS[®]) on the same day, they are reassessed in 24 hours. BioSIS[®] is an ocular disc made of porcine small intestine submucosa that is sutured to the cornea. If a perforation is very large, enucleation may be recommended.

Corneal perforations +/- iris prolapse

Call a veterinary ophthalmologist for referral. Before it is sent out the door, be sure to put on an Elizabethan collar and instill some atropine solution.

Indolent corneal ulcers

These are also called "Spontaneous Chronic Corneal Epithelial Defects" and do not heal in one week, with no identifiable underlying cause. There are "lipped" edges of the ulcer. Boxers, Corgis, and older patients are affected, along with Feline Herpes Virus cats. There is a deficiency of Substance P and a defect in epithelial adherence to the underlying stroma.

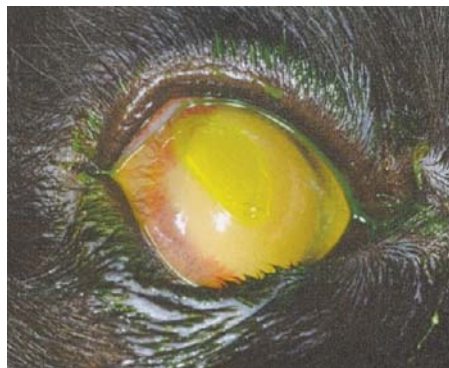


Fig 3: Indolent Corneal Ulcer


Therapy for indolent ulcers

Antibiotic choice is of little consequence as they are used to prevent infection. Antibiotics should be used TID, atropine SID and an oral NSAID for pain. A superficial keratectomy with a dry cotton tipped applicator and a grid keratotomy with a 27 g needle are the mainstays of treatment. Do not grid cats as this can increase the likelihood of corneal sequestrum formation. A canine soft contact lens acts as a splint or bandage to encourage adherence of the corneal layers and decreases pain. Using this therapy, healing often takes place within 2-4 weeks.

Corneal endothelial decompensation

Full thickness corneal edema results in bullae, or "blister," formation leading to ulcers that are very difficult to heal. Treat these as an indolent ulcer but avoid a grid keratotomy.

Summary

Determination of the cause of a corneal ulcer is the key to healing and working towards the best possible visual outcome. 

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