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Complicated Corneal Conditions

Introduction:

A sound knowledge of anatomy and function of the eye and its adnexa is important in understanding complex issues involving structures of the eye. The cornea is an avascular but metabolically active organ which functions to transmit light into the eye for further visual processing. The ability to maintain a clear cornea is dependent on not only proper corneal metabolism but on surrounding structures to function and protect this delicate but highly important structure.

Eyelid structure and function affects corneal health. Eyelids spread tear film and stabilize it by their many meibomian glands which produce the outer oily layer of the triple layer precorneal tear film. In addition proper eyelid function allows adequate spreading of the tear film over the ocular surface, provides protection from exposure, and reflexive action to protect the ocular surface from injury. Improper eyelid function can lead to insufficient spreading of the tear film resulting in tear film evaporation and corneal injury. Examples of disorders leading to evaporative tear film disorders include lagophthalmos, ectropion, facial nerve paralysis, etc.

Tear film and its constituents are essential in protecting the ocular surface. This triple layer consisting of an inner mucin layer derived from conjunctival goblet cells and corneal epithelial cell glycocalyx; the middle aqueous layer derived from lacrimal and third eyelid glands; and the oily layer layer as mentioned above from meibomian glands. This triple layer tear film not only flushes away debris, provides moisture, but is the major player in ocular surface immune defense. Without it the ocular surface becomes opaque, is prone to injury and invasion from environmental and local pathogens.

The cornea is comprised of three main layers but with other important constituents. This delicate structure still functions in a highly metabolic fashion but at the same time retaining relative dehydrated status in order to remain a clear refractive organ for light assembly into the eye. The outer epithelial layer is intimately associated with the tear film; the middle stromal layer with specially arranged collagen fibrils and very few keratocyte cells, and the inner endothelium (the

critical single layer of highly metabolically active cells mostly responsible for maintaining corneal deturgence). Injury or disease affecting any of these layers can result in permanent scarring and decreased vision. Diseases left ongoing without treatment will likely lead to permanent blindness.

The clinical relevance is that accurate identification of a problem involving the cornea is essential and will ultimately guide and direct treatment of the problem. The quicker the corneal injury can be fixed or the corneal disease or associated condition managed the better the outcome for corneal clarity and function. Keep in mind many of the discussed corneal conditions need long term therapy in order to maintain corneal health.

Corneal edema is characterized by swelling, thickening, and a blue haze to the cornea. This is incited by either damaged epithelium or endothelium. The damage may be temporary or permanent. Loss of the protective epithelium due to an ulcer or injury will result in imbibition of the tears into the stroma resulting in edema. In contrast, endothelial injury due to intraocular inflammation, elevated IOP, or primary endotheliopathy will also result in corneal edema because the pumping mechanism has been disrupted. This can be temporary as in elevated IOP or uveitis or can be permanent if the above are left untreated or with continued degeneration of the endothelial cells.

Neovascularization of the cornea is always abnormal. There is always a cause. The pattern and depth of neovascularization can signify where and what the inciting cause is. A patient with dorsal corneal neovascularization without an ulcer may be rubbing, may have eyelid disease or may have aberrant cilia (ectopic cilia, distichia, trichiasis). Deep vessels signify deep disease. Stromal ulcerative keratitis or uveitis are examples. Remember new vessels advance from the limbus and do so at a slow rate of approximately 1 mm per day. Once the underlying cause has been removed vessels regress often leaving ghost vessels which are easily revascularized with reinitiation of disease.

Corneal fibrosis is the end result of prior disease or injury. Degree of fibrosis will vary with degree of injury, longevity of disease. Although fibrosis is a natural consequence of corneal injury it reduces corneal clarity and ultimately vision and therefore careful tailoring of medications or selection of surgeries are essential to try and reduce the amount of fibrosis in our patients visual field. Judicious use of topical steroidal anti-inflammatories can reduce the formation and degree of fibrosis. In general cats corneas seem to recover from disease with less scar tissue than dogs.

Corneal pigmentation is common in ophthalmology patients and brachycephalic dog breeds make up the majority of patients seen for this condition. Other conditions associated with corneal melanosis and pigmentation include chronic superficial keratitis and keratoconjunctivitis sicca. The truth is not all patients who present with corneal pigmentation have KCS. So what causes pigmentation? In essence, anything leading to chronic irritation can result in corneal pigmentation. Pigment cells congregate at the limbus and surrounding conjunctiva and those breeds with heavier pigment in these areas seem to pigment more readily. Brachycephalic conformation plays a big part in formation of corneal pigment. These features include nasal trichiasis, medial canthal trichiasis, entropion, prominent globes, shallow orbits, lagophthalmos, decreased corneal sensitivity. The above features lead to evaporative tear film disorder and reduced overall protection of the corneal surface due to exposure resulting in chronically irritated corneal surface and laying down of not only pigment but neovascularization and fibrosis in severe cases. Therapy is best initiated before pigment progresses and is aimed at improving globe coverage with surgeries such as permanent canthoplasties, nasal fold resection, improving tear film and protecting the ocular surface with lubricants. Keratectomies and irradiation can be helpful in reducing the pigment but is often replaced with scar tissue and the long term benefit is only temporary.

Corneal ulcers are easily diagnosed with proper equipment, thorough evaluation of the ENTIRE corneal surface and fluorescein dye. However, the cause of the ulcer may not be as easily discerned. This may lead to recurrence of an ulcer, refractory ulcer, or worsening of the ulcer such that perforation of the cornea occurs. Tailoring your history and examination technique can provide insight into the cause of the ulcer. Location of the ulcer, degree of surrounding corneal edema, fibrosis (if any), neovascularization can give clues as to the cause of the ulcer as well as the longevity of the ulcer. Critical evaluation of the surrounding adnexa for foreign bodies, aberrant cilia, as well as thorough diagnostics including STT1 and IOP can assist in finding the cause of the ulcer. Superficial ulcers should heal in 7 days. Assuming the patient is wearing a protective e-collar and the client is being diligent with medications, an ulcer that has not healed in 7 days is considered non-healing. Re-examination for causes for the ulcer that may have been overlooked before is crucial. Not all patients with non-healing ulcers have SCCED. This is a middle-aged to older dog condition (aka Boxer ulcer) affecting the cornea and creating spontaneous ulcers that are always superficial with a characteristic lipping of the epithelium. Surgical keratotomy is almost always curative in cases that do not respond to simple cotton tipped debridement. These ulcers can recur and both eyes are predisposed. SCCED should not

be a high differential in a young dog with a non-healing ulcer. Other causes should be considered.

Complicated ulcers include stromal ulcers that are worsening, not healing, melting ulcers, and deep ulcers. Corneal abscesses though less common can become complicated quickly if proper treatment isn't initiated. Cytology and culture can help determine organism if septic. Therapy for these ulcers should not be taken lightly and these patients need to be seen frequently. Deep ulcers are best managed surgically in most cases and referral to an ophthalmologist should be strongly considered. Bacteria are the largest culprits of septic stromal ulcers and therapy should be broad-spectrum covering both gram positive and gram negative organisms keeping in mind that not all drugs penetrate well through the cornea. In particular abscesses without ulcer need treatment with antibiotics that penetrate through an intact epithelium. Organisms resistant to commonly used antibacterials are becoming increasingly common. Fungal keratitis is not as common and will often be overlooked without cytology. I see a few cases each year. Any sign of collagenolysis REQUIRES anticollagenolytic therapy. Sterile collection of autogenous serum, doxycycline, acetylcysteine, are some commonly available therapies used in the treatment of melting ulcers for their anti-collagenolytic properties. Our facility uses banked plasma mainly. Although acetylcysteine is widely available I rarely use it due to frequent complaints of severe ocular irritation upon application. Medical management needs to be aggressive and frequent in septic ulcers. Every one hour treatment round the clock and rechecking the next day. Don't forget the Elizabethan collar. Don't forget to treat the associated uveitis and pain. I usually use oral medications for these respectively. Surgical intervention is warranted in deep ulcers, worsening melting ulcers, descemetocelles, and perforations.

Immune-mediated corneal diseases include chronic superficial keratitis, punctate keratitis. These conditions are almost always bilateral and local immune-suppressants are recommended. Long term therapy is often necessary and recrudescence is expected if therapy is suspended. Other immune-mediated ocular or systemic conditions may be concurrent (ie. Immune-mediated KCS). Some patients require treatment with systemic immunosuppressants although in my experience this is rare.

Sometimes corneal changes are the first thing noticed by the owner and this precipitates an appointment with you the veterinarian. There are numerous conditions that cause secondary corneal pathology. It will be your challenge to determine whether the corneal changes are primary or secondary to some other condition or disease. Good examination technique, simple

diagnostics (STT, FS, IOP) and sound judgement will help facilitate appropriate treatment for the patient. We will take a look at various patients with corneal pathology and determine whether it is primary or secondary and go over therapies and prognosis.

Disease and conditions affecting the cornea are numerous and have not enough time to go over the entire list. There are excellent references that deserve a place on your bookshelf as well as resources available by phone and internet contact for the more challenging cases. Early referral to an ophthalmologist can improve outcome in these challenging cases and serve as continued education for you for future cases.