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Review on Multiple Diseases of Cornea in Dog and cat

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Summary: Multicourse result corneal disease of pet animals such as; corneal opacity, focal edema, diffuse edema, keratic precipitates, So, causes of corneal opacities, which are often seen in various combinations. Specific causes of focal edema include focal corneal epithelial dysfunction. The cornea is the clear, shiny membrane, which makes up the surface of the eyeball. Cornea is transparent, densely innervated, a vascular, and the major refractive structure of the eye. A healthy cornea achieves and maintains transparency due to the organization of constituent cells and collagen fibers, as well as its relatively dehydrated state. A complete ophthalmic examination is essential to identify related ocular and systemic abnormalities that may have led to the corneal opacity. Correct interpretation of corneal changes is critical for diagnosing corneal disease as well as many other ocular and some systemic diseases. Therefore, appropriate diagnostic tests and prompt initiation of optimal therapy are required to maximize the chance of saving vision, producing a comfortable globe and, occasionally, saving the patient's life. Treatment depends on whether there is a corneal abrasion, corneal ulcer, or descemetocele present. Corneal abrasions generally heal within 7-10 days. Medication is used to prevent bacterial infections and to relieve pain. Antibiotic drops are only effective for a few minutes so they must be applied frequently; ointments last a bit longer but still require application every few hours. It is suggested that an antibiotic preparation be instilled in the eye 4 to 6 times per day.
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Key words: Cornea, disease, edema and ulcer

1. INTRODUCTION

The cornea is transparent, densely innervated, a vascular, and the major refractive structure of the eye. A healthy cornea achieves and maintains transparency due to the organization of constituent cells and collagen fibers, as well as its relatively dehydrated state. Anything that alters this organization or deturgescence leads to development of a corneal opacity.1 as the outer and most exposed structure of the globe, the cornea is not only at risk for trauma, but also often receives close attention from clients. As such, a presenting complaint of corneal opacity is common in veterinary practice (Torricelli and Wilson, 2014).

Correct interpretation of corneal changes is critical for diagnosing corneal disease as well as many other ocular and some systemic diseases. Therefore, appropriate diagnostic tests and prompt initiation of optimal therapy are required to maximize the chance of saving vision, producing a comfortable globe and, occasionally, saving the patient's life (Gelatt *et al*., 2013).

2. CORNEAL OPACITY

2.1. Causes

Causes of corneal opacities, which are often seen in various combinations, are listed in Table 1.2 each of these pathologic changes is associated with a specific appearance especially color. However, assessment of texture, depth, location, pattern, shape, and outline of the region of opacifi cation isalso important as some colors can look similar (Townsend, 2008).

TABLE 1.Causes of Corneal Opacity&Appearance		
CAUSES OF CORNEAL OPACITY		CORNEAL APPEARANCE
Edema		Blue "cobblestoned" appearance
Inflammatory cell infiltration		Yellow, green, or tan corneal stromal opacity
Lipid/mineral deposition		Silvery white, crystalline, sparkly opacities; sometimes coalescing creamy or shiny opacities
Fibrosis		Grayish white, sometimes feathery or wispy opacity
Melanosis		Dark brown to black, variable density; often with blood vessels
Vascularization		Variably perfused blood vessels extending from corneoscleral limbus

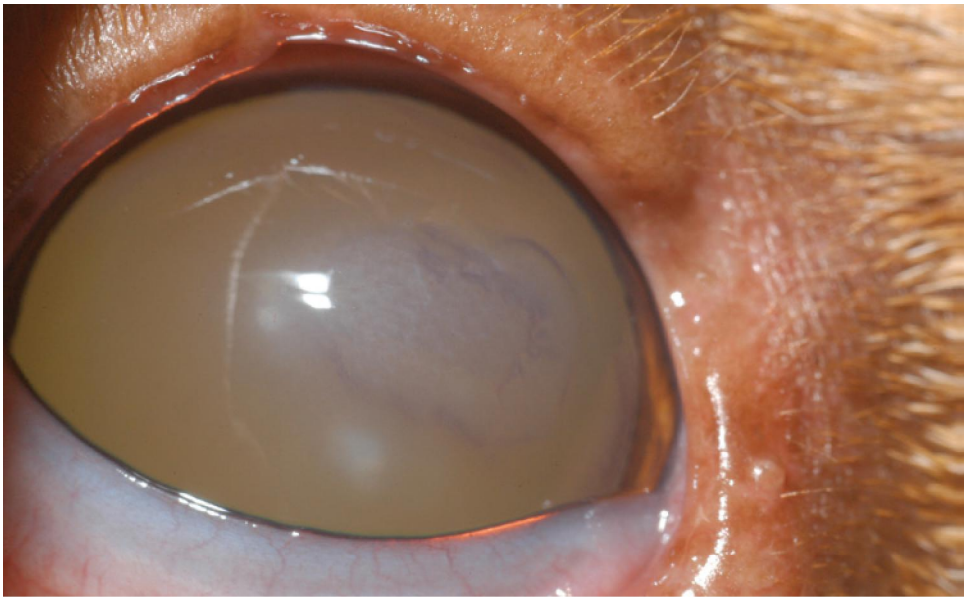

Figure: 1. Focal corneal edema secondary to corneal ulceration in a dog. The pupil has been pharmacologically dilated. Note the loose epithelial edges of the corneal ulcer suggestive of a superficial chronic corneal epithelial defect (SCCED) (Moore, 2005). Resolution was achieved by debridement (using a sterile cotton-tipped applicator) and topically applied antibiotics and atropine. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service)*

2.2. Edema

A blue, "cobblestoned" appearance of the cornea whether focal or diffuse is indicative of corneal stromal edema, and results from loss of corneal epithelial or endothelial cell function.

2.2.1. Focal Edema:

Specific causes of focal edema include focal corneal epithelial dysfunction (due to corneal ulceration), or focal endothelial cell damage (due to blunt corneal trauma, lens luxation, or keratic precipitates) (Figure: 1).

2.2.2. Diffuse Edema.

Diffuse corneal edema (Figure 2) is more consistent with intraocular disease, such as glaucoma or uveitis, but may also indicate breed-related endothelial dystrophy or age-related endothelial degeneration. These conditions can usually be differentiated by assessing for signs of pain or inflammation: the latter 2 conditions are non-painful and non-inflammatory, whereas glaucoma and uveitis are painful and associated with other signs of inflammation (Spies *et al*., 2009).

Whenever possible, treatment for diffuse corneal edema should always be directed at the underlying disease process, especially since both glaucoma and uveitis are vision-threatening diseases, and some causes of uveitis can even be life threatening. When no cause is found or the underlying disease is untreatable, symptomatic treatment with hypertonic sodium chloride (Nacl) ophthalmic ointment may be attempted, but it often has little to no effect, may irritate the eye, and requires administration at least 4 to 6 times daily.

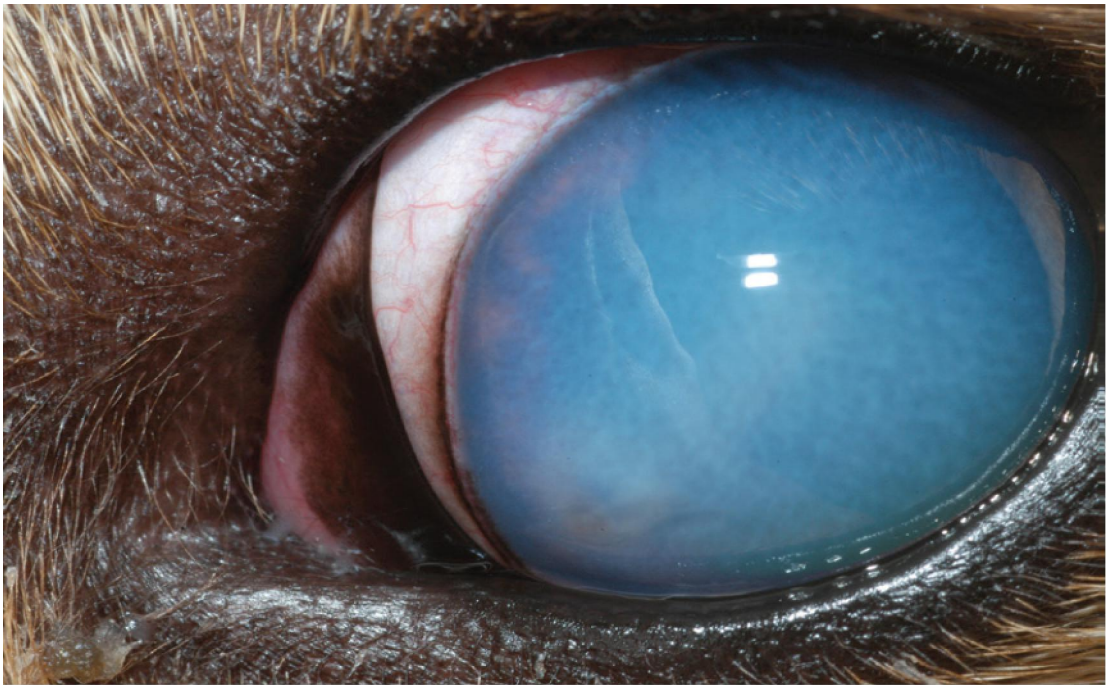

Figure: 2. Diffuse corneal edema in a dog with a penetrating corneal cat claw injury and secondary uveitis. The wound from the cat claw can be seen on the ventromedial paraxial cornea. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service).*

2.2.3. Diffuse Edema in Cats.

Cats are rarely affected with endothelial dystrophy or senile endothelial degeneration. instead, severe, diffuse corneal edema in cats is typically seen with acute bullous keratopathy (Figure 3). This condition, which is more common in younger cats, has no recognized predisposing cause and an extremely acute onset that leads to massive stromal edema and, sometimes, corneal rupture. The dysfunction may involve the stroma itself rather than the endothelium or epithelium. Treatment involves emergency surgical support—either conjunctival grafting or placement of a third eyelid flap (Spies *et al*., 2009).

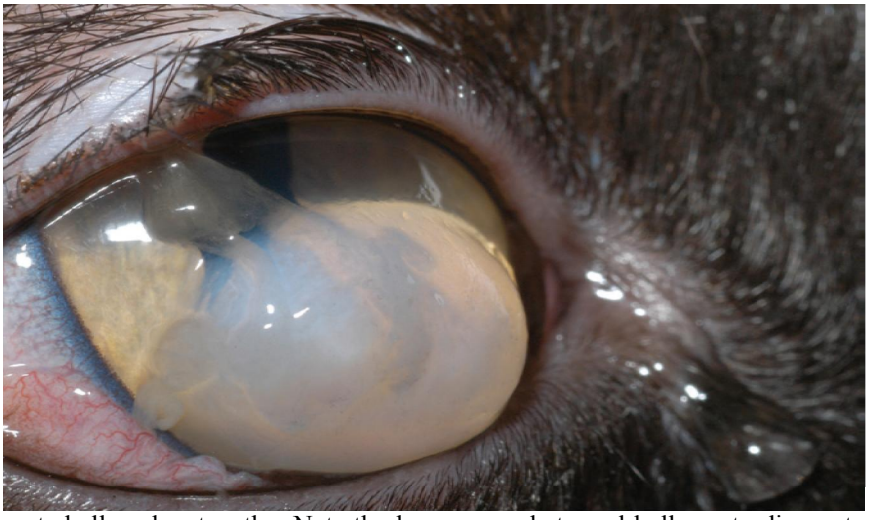

Figure: 3. Feline acute bullous keratopathy. Note the large corneal stromal bulla protruding outwards. The lack of corneal vascularization confirms the acute nature of the disease. Mucous discharge covers some of the dorsomedial cornea and bulla. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service)*

2.3. Inflammatory Cell Infiltration

A yellow, green, or tan corneal stromal opacity suggests white blood cell (WBC) infiltration, which conjunctival grafts surgery, is often recommended to provide immediate tectonic support and promotes healings (Spies *et al*., 2009).

In some disease processes infiltration of the corneal stroma by WBCs is evident as excrescences above the corneal surface examples include feline eosinophilic keratitis (FeK; Figure 5) and chronic superficial keratitis (csK, or "pannus"; Figure 6) in dogs, or neoplastic keratitis (typically lymphoma). In these instances, the cornea typically becomes heavily vascularized due to chronicity of the process. For the immune-mediated keratitides, it is important to treat any underlying conditions.

For example, feline herpesvirus type 1 (FhV-1) is believed to cause FeK in many cats; in dogs, ultraviolet (UV) light exposure is a risk factor for development and progression of csK. These immune-mediated diseases must be controlled with topical application of steroids and/or calcineurin inhibitors, such as cyclosporine or tacrolimus (Spies *et al*., 2009).

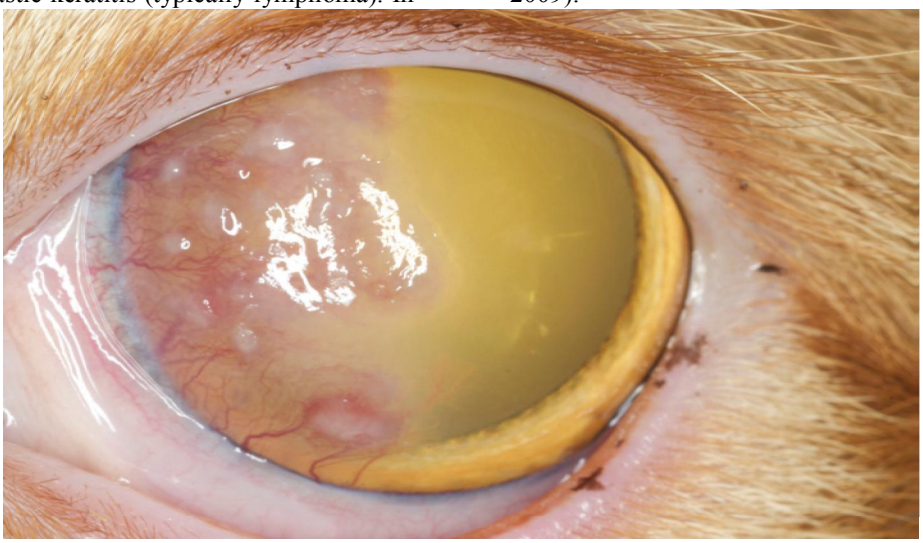

Figure: 5. Feline eosinophilic keratitis. Note the dense superficial corneal vessels and raised white corneal plaques. Diagnosis is made by cytology, and treatment consists of antiviral and immunomodulatory drugs. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service)*

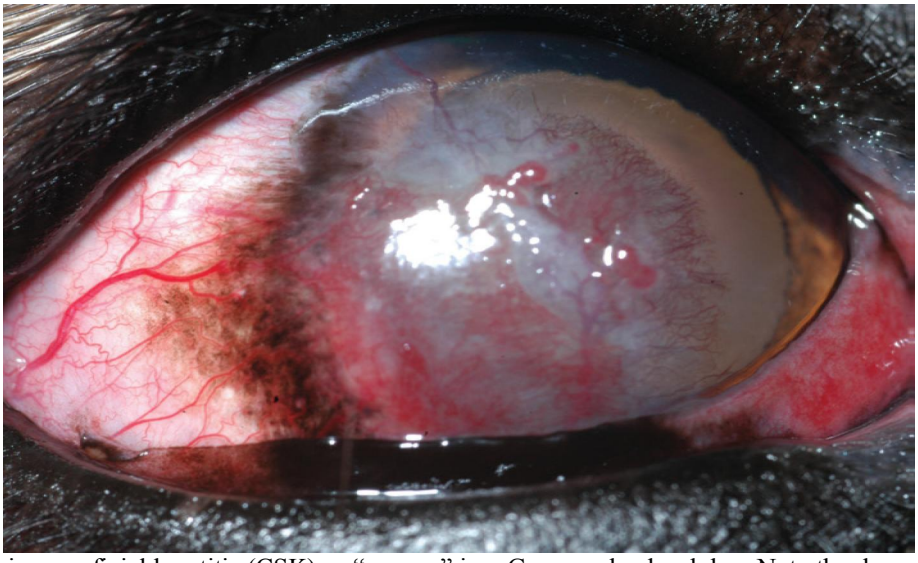

Figure: 6. chronic superficial keratitis (CSK) or "pannus" in a German shepherd dog. Note the dense lateral paraxial corneal plaque. This is an immune-mediated condition in which ultraviolet (UV) light exposure is a cofactor. Treatment includes topical immunomodulatory drugs and decreased UV exposure. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service).*

2.4. Keratic Precipitates.

White blood cells (WBC), typically in association with fibrin, can also form keratic precipitates (KPs)—more discrete clumps located on the inner corneal endothelium that appear as focal tan spots (sometimes likened to mutton fat because of their greasy appearance; Figure 7). Due to most frequently caused by bacterial infection of the cornea, often *Pseudomonas* or beta-hemolytic *Streptococcus* species (Figure 4) (Barrientos *et al*., 2013).

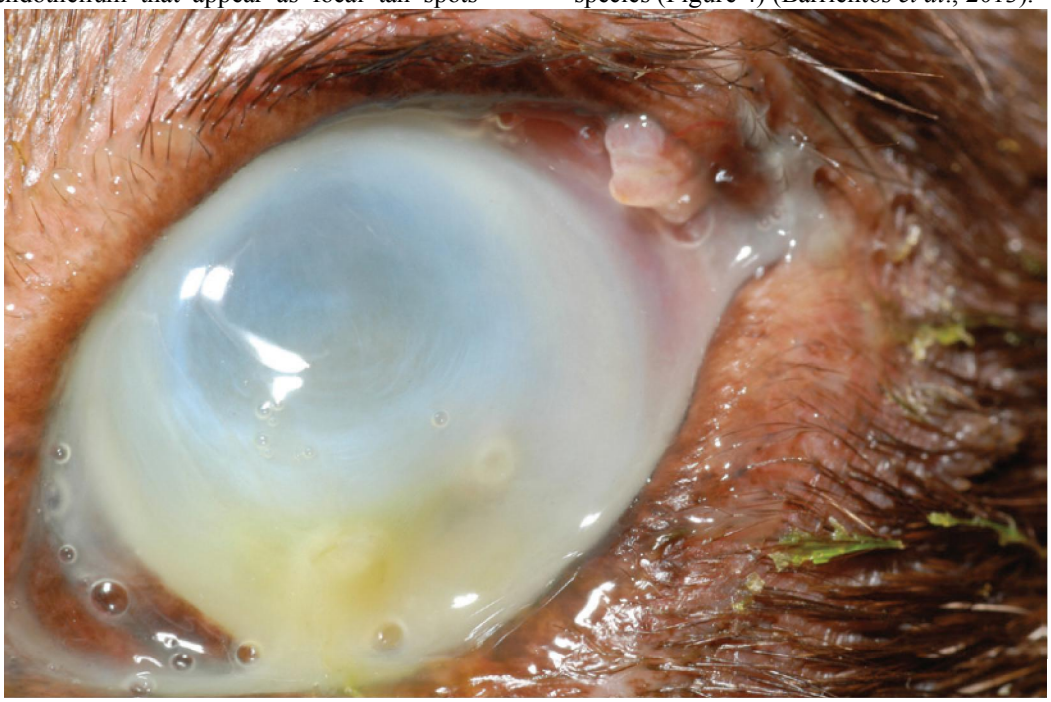

Figure: 4. Infectious keratitis with keratomalacia ("melting") and stromal loss in the dogs. The patient also has an eyelid tumor on the lateral aspect of the superior eyelid. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service).*

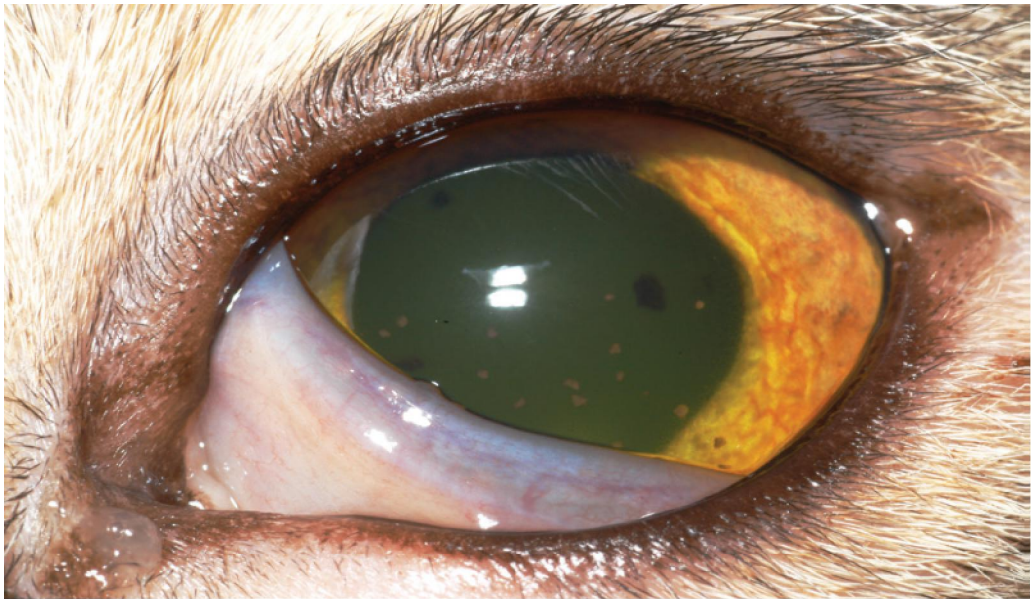

Figure: 7. Keratic precipitates (KPs) in a cat with uveitis. Rubeosis iridis (neovascularization of the iris) is also present. The brown KPs are located on the inner aspect of the corneal endothelium, predominantly ventrally. KPs are a pathognomonic sign of anterior uveitis. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service)*

2.5. Fibrosis

If the cornea has a grayish white, sometimes feathery or wispy opacity, it is most likely fibrosed (Figure 8). Hypo perfused (or "ghost") corneal blood vessels may be seen in association with resolving or inactive corneal fibrosis, but well perfuse vessels indicate more active keratitis.

In most cases, corneal scarring is permanent, but can decrease over time if the underlying cause of corneal damage is removed, especially in younger animals. Corneal scars do not retain fluorescein stain and require no further treatment. However, if extensive scarring cause's vision loss, corneal transplantation may beconsidered.

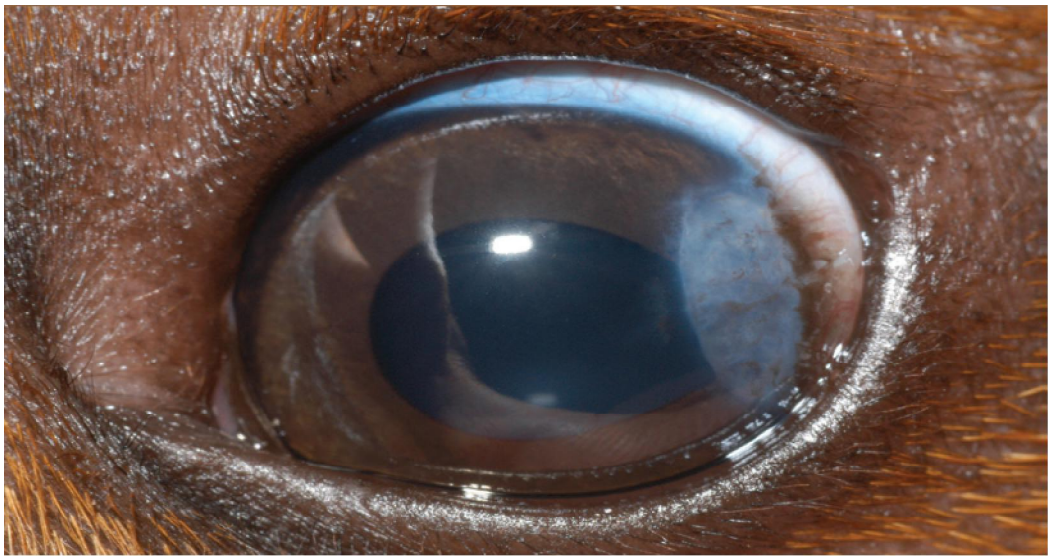

Figure: 8. Focal corneal fibrosis in a dog. Note the gray appearance and crisp edges of the lesion. Some superficial corneal blood vessels are also apparent. This lesion has minimal effect on vision and does not require any treatment. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service).*

2.6. Lipid/Mineral Deposition

Silvery white, crystalline, sparkly opacities (or sometimes coalescing creamy or shiny opacities) typically located in the anterior stromal, immediately under the corneal epithelium, represent lipid (typically cholesterol) or mineral (typically calcium) deposits.9 these can: 2, 10, Occur secondary to chronic keratitis or senile degeneration (also known as corneal or calcareous degeneration or calcific band keratopathy (Wang *et al*., 2008).

2.7. Location of corneal opacity

Accurate judgment of lesion depth within the cornea requires use of magnification and a slit lamp; however, corneal vessels can also be useful clinical guides:

• *Superficial corneal blood vessels* arise from the conjunctiva at the limbus and are fine, branching.
• *Vessels that arise from under or within the scleral shelf* are located deeper in the cornea, where they form a more "hedge-shaped" pattern (Figure 9). They are characteristic of deeper, more serious, vision-threatening diseases, such as deep keratitis, uveitis, and glaucoma.

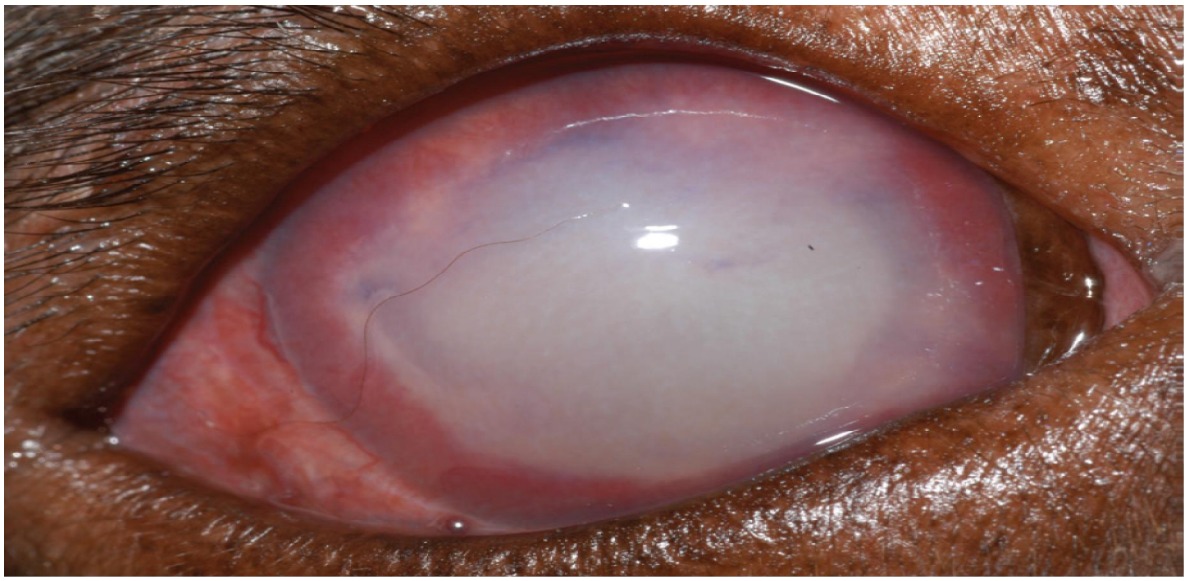

Figure: 9. deep corneal vessels in a dog with uveitis and glaucoma. Note that the vessels are "hedge-like." In this patient, vessels were present for 360 degrees around the limbus. Deep corneal vessels should stimulate a thorough intraocular examination, including measurement of intraocular pressure. (Source: *Courtesy University of California–Davis Comparative Ophthalmology Service).*

TABLE 2. Location & Cause of Corneal Opacities

CORNEAL OPACITY LOCATION		LIKELY CAUSE & RECOMMENDATIONS
Paraxial corneal ulcer		Adnexal region closest to corneal ulcer should be investigated for abnormalities, such as distichia, ectopic cilia, or foreign bodies (often associated with intense blepharospasm), or entropion/ectropion.
Lateral perilimbal to paraxial corneal edema of both eyes		Typical of age-related corneal endothelial degeneration; edema Progresses medially until it covers entire cornea causing vision impairment.
Circumferential perilimbal corneal edema, cellular infi ltrates, and/or vessels		Can indicate diffuse episcleritis or lymphoma2
Corneal ulcer and focal edema near 12 o'clock position on dorsal paraxial Cornea.		In a young dog, warrants thorough examination for ectopic cilia (certain breeds, such as Shih Tzu, are predisposed)
Diffuse corneal edema		Typically indicates intraocular disease (Figure 2)
Diffuse superficial corneal vessels		Suggests chronic, diffuse superfi cial irritation, such as keratoconjunctivitis sicca (KCS or *dry eye*); when associated with mucoid to mucopurulent ocular discharge, a Schirmer's tear test (STT) should be performed
Focal corneal edema		Often indicates corneal surface (epithelial) disease (Figure 1).
Lateral perilimbal cornea; raised Plaque		Most common location for dense cellular infi ltrate and corneal vascularization seen in CSK and FEK; in more advanced cases, will spread medially

2.8. Diagnosis

2.8.1. Ophthalmic Examination:

A complete ophthalmic examination (and general physical examination, if systemic causes are suspected) is essential to identify related ocular and systemic abnormalities that may have led to the corneal opacity. In particular, anatomic or functional eyelid abnormalities, such as lagophthalmos, distichiasis, eyelid tumors, blepharitis, facial nerve paralysis, or conjunctival foreign bodies, should be carefully ruled in or out (Maggs *et al*., 2013).

2.8.1.1. Ophthalmic Diagnostic Tests

A) Tonometry & Ultrasound: diffuse corneal edema, deep corneal vessels, or episcleral injection are each indications of intraocular disease, and tonometry (measurement of intraocular pressure) and ocular ultrasound (if a complete intraocular examination is not possible) are recommended to assess for evidence of glaucoma, uveitis, intraocular masses, retinal detachment, or lens luxation.

B) Schirmer's Tear Test. Presence of conjunctivitis and mucoid discharge combined with superficial corneal blood vessels, fibrosis, or melanin

(in various combinations) are strongly suggestive of Kcs, and a schirmer's tear test (sTT) should always be performed.

C) Palpebral Reflex:an absent or decreased palpebral reflex predisposes to corneal exposure; a full neurologic examination, especially of cranial nerves, is warranted. Any patient with corneal opacity should have fluorescein stain applied topically to assess for corneal ulceration. This evaluation should be performed after all other assessments are finished since it masks many other examination findings, and affects results of other tests, such as the sTT, corneal cultures, and cytology (Maggs *et al*., 2013).

2.8.2. Cytology

Samples for cytologic assessment obtained using the blunt end of a scalpel blade, Kimura spatula, or cytobrush and aerobic bacterial ± fungal culture are excellent means to differentiate immune-mediated from infectious processes, and form the minimum database for investigation of corneal disease (Sansom and Blunden, 2010).

2.9. Occurrence of corneal opacity

Naturally, history is one of the most valuable tools to establish the duration of a corneal opacity, but other clues are available through examination. For example:1) *Acute corneal opacities* (< 3–5 days) do not have corneal vessels unless the vessels were present prior to the current lesion. it is estimated that corneal vessels first appear approximately 4 days after the initial corneal injury; then grow approximately 1 mm per day. 2) *Active lesions* usually have more irregular, or *fuzzy*, outlines due to corneal edema and/or cellular infiltrates. 3) *Chronic, inactive lesions*, such as corneal fibrosis or melanosis, often have more distinct borders with the surrounding cornea.

2.10. Determining Whether a Corneal Ulcer Is Complicated

A corneal ulcer is considered complicated if one or more of the following is noted:Corneal blood vessels, Corneal stromal loss (If > 50% is lost, surgical stabilization is typically recommended.), Corneal WBC infiltration (greenish yellow cornea), Failure to heal within 1 week, Indolent ulcers (ie, SCCEDs), Infection, Keratomalacia ("melting") and Marked or persistent corneal edema.

2.11. Treatment

Table: 3. Treatment of corneal opacity with respect to it lesion.

Lesions	Treatments
Yellow/green (WBCs)	Treat with anti virals or anti-inflammatories Intensively treat with topical antimicrobials. (Guided by cytology and C/S results). If keratomalacia present, use serum topically. If > 50% stromal loss, consider surgical stabilization.
Blue (stromal edema)	Consider treating with hypertonic (5%) NaCl ointment ≥ Q 6 H. Treat with prophylactic topical antibiotics and atropine
Creamy or sparkly white (lipid/mineral)	Treat underlying systemic disease and/ or primary keratitis. Monitor for ulceration
Gray (fibrosis)	No treatment required unless underlying cause found

3. CORNEAL ULCER

The cornea is the clear, shiny membrane, which makes up the surface of the eyeball. It is much like a clear window. To understand a corneal ulcer, you must first understand how the cornea is constructed. The cornea is comprised of three layers.

The most superficial layer is the epithelium. Actually, this layer is comprised of many, very thin layers of cells. Below the epithelium is the stromal, and the deepest layer is Descemet's membrane. Because all of these layers are clear, it is not possible to see them without special stains and a microscope.

3.1. Definition

Erosion through a few layers of the epithelium is called ***corneal erosion*** or a *corneal abrasion*. **A *corneal ulcer*** is erosion through the entire epithelium and into the stromal. If the erosion goes through the epithelium and stromal to the level of Descemet's membrane, a *descemetocele* exists. If Descemet's membrane ruptures, the liquid inside the eyeball leaks out and the eye collapses.

3.2. Etiology

There are several causes for corneal ulcers in dogs. The most common is trauma. An ulcer may result from blunt trauma, such as a dog rubbing its eye on carpet, or due to a laceration, such as a cat scratch. The second most common cause is chemical burn of the cornea. This may happen when irritating shampoo or dip gets in the eye (Spies *et al*., 2009).

Less common causes of corneal ulcers include bacterial infections, viral infections, and other diseases. These may originate in the eye or develop secondary to disease elsewhere in the body. Examples of other diseases include Epithelial Dystrophy (a softening of the cornea which is inherited in breeds such as the Boxer), Keratoconjunctivitis Sicca (drying of the cornea due to abnormal tear formation), and diseases of the endocrine system (diabetes mellitus, hyperadrenocorticism, and hypothyroidism) (Maggs *et al*., 2013).

3.3. Types of corneal ulcer:

3.3.1. Superficial and deep corneal ulcers

Corneal ulcers are a common human eye disease. They are caused by trauma, particularly with vegetable matter, as well as chemical injury, contact lenses and infections. Other eye conditions can cause corneal ulcers, such as entropion, distichiasis, corneal dystrophy, and keratoconjunctivitis sicca (dry eye). Many micro-organisms cause infective corneal ulcer. Among them are bacteria, fungi, viruses, protozoa, and Chlamydia (Glover *et al*., 1994).

Bacterial keratitis is caused by Staphylococcus aureus, Streptococcus viridans, Escherichia coli, Enterococci, Pseudomonas, Nocardia, N. Gonorrhoea and many other bacteria.

Fungal keratitis causes deep and severe corneal ulcer. It is caused by Aspergillus sp., Fusarium sp. Candida sp., as also Rhizopus, Mucor, and other fungi. The typical feature of fungal keratitis is slow onset and gradual progression, where signs are much more than the symptoms. Small satellite lesions around the ulcer are a common feature of fungal keratitis and hypopyon is usually seen (Spies *et al*., 2009).

Viral keratitis causes corneal ulceration. It is caused most commonly by Herpes simplex, Herpes zoster and Adenoviruses. Also it can be caused by corona viruses & many other viruses. Herpes virus causes a dendritic ulcer, which can recur and relapse over the lifetime of an individual.

Protozoa infection like Acanthamoeba keratitis is characterized by severe pain and is associated with contact lens users swimming in pools.

Chlamydiae trachomatis can also contribute to development of corneal ulcer. Superficial ulcers involve a loss of part of the epithelium. Deep ulcers extend into or through the stroma and can result in severe scarring and corneal perforation. Descemetoceles occur when the ulcer extends through the stroma. This type of ulcer is especially dangerous and can rapidly result in corneal perforation, if not treated in time (Maggs *et al*., 2013).

The location of the ulcer depends somewhat on the cause. Central ulcers are typically caused by trauma, dry eye, or exposure from facial nerve paralysis or exophthalmos. Entropion, severe dry eye and trichiasis (inturning of eyelashes) may cause ulceration of the peripheral cornea. Immune-mediated eye disease can cause ulcers at the border of the cornea and sclera. These include Rheumatoid arthritis, rosacea, systemic scleroses which lead to a special type of corneal ulcer called Mooren's ulcer. It has a circumferential crater like depression of the cornea, just inside the limbus, usually with an overhanging edge (Spies *et al*., 2009).

3.3.2. Refractory corneal ulcers

Refractory corneal ulcers are superficial ulcers that heal poorly and tend to recur. They are also known as indolent ulcers or Boxer ulcers. They are believed to be caused by a defect in the basement membrane and a lack of hemidesmosomal attachments. They are recognized by undermined epithelium that surrounds the ulcer and easily peels back. Refractory corneal ulcers are most commonly seen in diabetics and often occur in the other eye later. They are similar to Cogan's cystic dystrophy.

3.3.3. Melting ulcers

Melting ulcers are a type of corneal ulcer involving progressive loss of stroma in a dissolving fashion. This is most commonly seen in

Pseudomonas infection, but it can be caused by other types of bacteria or fungi. These infectious agents produce proteases and collagenases which break down the corneal stroma. Complete loss of the stroma can occur within 24 hours. Treatment includes antibiotics and collagenase inhibitors such as acetylcysteine. Surgery in the form of corneal transplantation (penetrating keratoplasty) is usually necessary to save the eye.

3.3.4. Corneal healing

An ulcer of the cornea heals by two methods: migration of surrounding epithelial cells followed by mitosis (dividing) of the cells, and introduction of blood vessels from the conjunctiva. Superficial small ulcers heal rapidly by the first method. However, larger or deeper ulcers often require the presence of blood vessels to supply inflammatory cells. White blood cells and fibroblasts produce granulation tissue and then scar tissue, effectively healing the cornea.

3.4. Clinical sign

Animals with ulcerated corneas exhibit blepharo spasm, epiphora, and often are photophobic. These animals also may appear head shy and reluctant to allow physical examination of the head region.

Corneal ulcers are extremely painful due to nerve exposure, and can cause tearing, squinting, and vision loss of the eye. There may also be signs of anterior uveitis, such as miosis (small pupil), aqueous flare (protein in the aqueous humour), and redness of the eye. An axon reflex may be responsible for uveitis formation stimulation of pain receptors in the cornea results in release inflammatory mediators such as prostaglandins, histamine, and acetylcholine. Sensitivity to light is also a common symptom of corneal ulcer (Barrientos *et al*., 2013).

3.5. Pathogenesis

A corneal ulcer is painful. In response to pain, most dogs rub the affected eye with a foot or on the carpet. To protect the eye, they keep the lids tightly closed. Occasionally, there will be a discharge that collects in the corner of the eye or runs down the face.

3.6. Diagnosis

Superficial corneal abrasions are usually not visible. They can be visualized with the use of fluorescein stain. A drop of this stain is placed on the cornea. The dye will adhere to an area of ulceration and is easily visualized with a special black light called a Wood's light. This is the most basic test performed and may be the only test needed if the ulcer is acute and very superficial. If the ulcerated area is chronic or very deep, samples are taken for culture and cell study prior to applying the stain or any other medication (Barrientos *et al*., 2013).

Diagnosis is done by direct observation under magnified view of slit lamp revealing the ulcer on the cornea. The uses of fluorescein stain, which is taken up by exposed corneal stroma and appears green, helps in defining the margins of the corneal ulcer, and can reveal additional details of the surrounding epithelium. Herpes simplex ulcers show a typical dendritic pattern of staining. Rose-Bengal dye is also used for supra-vital staining purposes, but it may be very irritating to the eyes. In descemetoceles, the Descemet's membrane will bulge forward and after staining will appear as a dark circle with a green boundary, because it does not absorb the stain. Doing a corneal scraping and examining under the microscope with stains like Gram's and KOH preparation may reveal the bacteria and fungi respectively.

3.7. Treatment

Treatment depends on whether there is a corneal abrasion, corneal ulcer, or descemetocele present. Corneal abrasions generally heal within 7-10 days. Medication is used to prevent bacterial infections (antibiotic ophthalmic drops or ointment) and to relieve pain (atropine ophthalmic drops or ointment) (Barrientos *et al*., 2013).

Antibiotic drops are only effective for a few minutes so they must be applied frequently; ointments last a bit longer but still require application every few hours. It is suggested that an antibiotic preparation be instilled in the eye 4 to 6 times per day. On the other hand, the effects of atropine last many hours so this drug is only used once to twice daily.

If a corneal ulcer or descemetocele is present, measures must be taken to protect the eye and to promote healing. Since dogs do not wear eye patches well, surgical techniques are often used to close the eyelids and cover the ulcer or descemetocele. These measures protect the eye for several days, and then are reversed so the dog can use the eye again.

Ulcers that do not heal well often have a buildup of dead cells at the ulcer edge. These dead cells prevent normal cells from the corneal surface from sliding over the ulcer edge and filling in the defect. If this appears to be part of the healing problem, the dead cells are removed from the edges of the ulcer before the eyelids are surgically closed. In some cases, removing the dead cells may be all that is needed to start the healing process, so surgical

closing of the eyelids may not be necessary (Barrientos *et al*., 2013).

3.8. Side effect of treatment

Rarely, a dog will be allergic to an antibiotic that is instilled in the eye. If your dog seems to be in more pain after the medication is used, discontinue it and contact the veterinarian.

A dog with a corneal ulcer has quite a bit of pain in the eye, so it keeps it tightly shut. Atropine is used to relieve that pain. However, atropine also dilates the pupil widely. This means that the dog is very sensitive to light in that eye. Because of the light sensitivity, the eye will be held closed in bright light.

Atropine's effects may last for several days after the drug is discontinued. Do not be alarmed if the pupil stays dilated for several days. Should you accidentally get atropine in your eye; the same prolonged pupillary dilation will occur (Spies *et al*., 2009).

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REFERENCES

[1]. Barrientos, L.S, Zapata, G.and Crespi, J.a. (2013). a study of the association between chronic superficial keratitis and polymorphisms in the upstream regulatory regions of dla-drb1, dla-dQb1 and dla-dQa1. *Vet Immunol Immunopathol*; 156(3-4):205-210.

[2]. Gelatt, K.N., Gilger, B.C. and Kern, T.J. (2013). *Veterinary Ophthalmology*, 5th ed. Wiley blackwell, (eds).

[3]. Glover, T. Nasisse, M.P. and Davidson, M.G. (1994). Acute bullous keratopathy in the cat. *Vet Comp Ophthalmol*; 4(2):66-70.

[4]. Maggs, D.J, Miller, P. and Ofri, R. (2013). *Slatter's Fundamentals of Veterinary Ophthalmology*, 5th ed. elsevier.

[5]. Moore, P.A. (2005). Feline corneal disease. *Clin Tech Small Anim Pract*; 20(2):83-93.

[6]. Sansom, J. and Blunden, T. (2010). Calcareous degeneration of the canine cornea. *Vet Ophthalmol*; 13(4):238-243.

[7]. Spies, A.K., sapienza, J.S. and Mayordomo, A. (2009). Treatment of proliferative feline eosinophilic keratitis with topical 1.5% cyclosporine: 35 cases. *Vet Ophthalmol*; 12(2):132-137.

[8]. Torricelli, A.A. and Wilson, S.E. (2014). Cellular and extracellular matrix modulation of corneal stromal opacity. *Exp Eye Res,* 129c:151-160.

[9]. Townsend, W.M. (2008).Canine and feline uveitis. *Vet Clin North Am Small Anim Pract*; 38(2):323-346, vii.

[10]. Wang, l, Pan, Q. and Xue, Q. (2008). Evaluation of matrix metalloproteinase concentrations in precorneal tear fi lm from dogs with Pseudomonas aeruginosa-associated keratitis. *Am J Vet Res*; 69(10):1341-1345.

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