Corneal disease and treatment

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A starting point, when assessing corneal disease, is to appreciate corneal anatomy – it has an epithelium with a basement membrane, beneath which is the stromal layer, Descemet's membrane and the endothelial layer. The intregrity of the layers is vital to the health of the cornea. It is maintained in a 40% dehydrated state protected by the functional intregrity of the outer and inner membranes namely the epithelium and the endothelium. A failure of these layers permits the ingress of fluid and the hydration of the tissues leading to corneal oedema and loss of transparency.

CLINICAL ASSESSMENT

In making a clinical assessment one assesses the following:

- Previous history;
- Eyelid anatomy;
- Facial anatomy/globe size;
- Tear production quality and quantity;
- Breed;
- Clinical disorders; and
- Contralateral eye.

One notes the facial anatomy (see Figure 1) including the eyelid anatomy (see Figures 2 and 3).



Figure 1: Facial anatomy - lagophthalmos.



Figure 2: Entropian.



Figure 3: Ectropian.

The ultimate aid is the slit lamp biomicroscope (see Figure 4) but a good-quality magnifying loupe (see Figure 5) x2.5-4 times magnification is a very good aid in accurate diagnosis.



Figure 4: The slit-lamp biomicroscope.



Figure 5: Binocular magnifying loupe.

COLOUR CHANGES

One also notes colour changes as a guide to direct diagnosis. If the cornea is white, this indicates oedema, lipid,

scarring and infectious agents. If it is red, neovascularisation, stromal haemorrrhage, symblepharon and neoplasia, are suspected. And, if the cornea shows pigmented lesions, this can mean there is superficial benign melanin, sequestreum, or deep uveal pigment (see Figures 6, 7 and 8).



Figure 6: Oedema is indicated.



Figure 7: Neovascularisation in the eye.



Figure 8. Corneal sequestrum.

NON-INFLAMMATORY LESIONS

Corneal disease can be categorised into non-inflammatory lesions (see Figures 9, 10, 11, 12), which are divided into congenital non-inflammatory lesions (persistant pupillary membranes) and acquired non-inflammatory lesions (lipid keratopathy, band keratopathy and corneal endothelial dystrophy; and inflammatory lesions.



Figure 9: Persistent pupillary membranes with corneal lesions.



Figure 10: Lipid keratopathy.



Figure 11: Corneal lipid dystrophy.



Figure 12: Corneal endothelial dystrophy.

INFLAMMATORY LESIONS

Inflammatory lesions can be ulcerative or non-ulcerative. Ulcerative inflammatory disease can consist of:

- Physical;
- Chemical;
- Infectious;
- Dry eye;
- Neurotropic;
- Immunological; and
- Metabolic.



Figure 13: Dry eye.

Non-ulcerative inflammatory diseases consist of superficial keratitis, which can be mechanical (hairs, foreign bodies); infectious (bacterial, fungal, *Leishmania pannus*; eosinophilic keratitis; pigmentary keratitis; and herpesvirus keratitis.

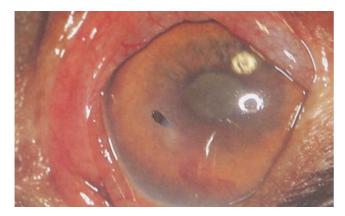


Figure 14: Corneal foreign body.

NORMAL CORNEAL HEALING AND CORNEAL STABILITY

Before discussing ulcerative corneal disease, we should remind ourselves of normal corneal healing and the considerations in the assessment of corneal stability. The corneal epithelium has great regenerative capacity. It responds within minutes to injury and can totally reepithelialise within four to seven days.

The cornea stroma heals by two mechanisms:

- Avascular healing resting keratocytes activate into fibroblasts and synthesise collagen; and
- Vascular healing extensive cell invasion, blood vessels invade, and fibrovasculargranulation tissue is laid down to form a dense scar.

Stromal healing is slow and often the epithelium regrows leaving a stromal deficit which then slowly fills.

Corneal assessment includes assessing the facial/eyelid anatomy; trichaiasis/distichiasis/ectopic cilia; lid function – blink ability, paresis/paralysis; tear production – quantity: the Schirmer tear test; tear quality – tear-film break-up time; and exposure to dryness.

PRE-CORNEAL TEAR FILM

The first layer to consider in assessing the cornea is the

tear film (see Figure 15). It is also important to make this assessment before other tests are performed involving the corneal surface.

Traditionally, one tends to consider the aqueous aspect of the composition of tears (Schirmer tear test), but one needs to be aware of the mucin and lipid composition also.

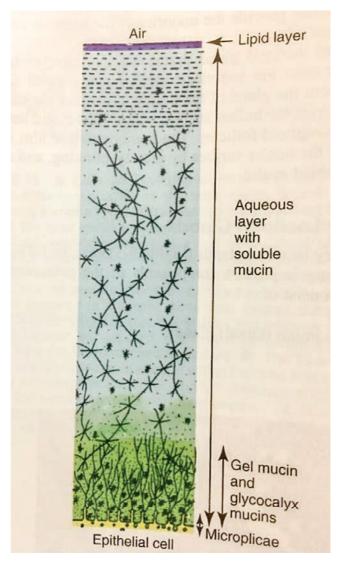


Figure 15: Pre-corneal tear film.

BASIC TESTS

Basic tests include the Schirmer tear test (see Figure 16); the Rose bengal stain; and the fluorescein stain. You must also assess the corneal sensitivity (nerve function).



Figure 16: Schirmer tear test.

CORNEAL ULCERATION AND NEW TREATMENT OPTIONS

Corneal ulceration is probably the category most clinicians consider when considering corneal disease. One must define the type and, if possible, the cause of the corneal ulcer. Then one must consider the treatment options available – supportive, medical, surgical, iontophoresis, and corneal-cross linkage.

SUPPORTIVE

Keep facial hair under control and keep cornea moist (OptixcareEye Plus tid).

MEDICAL

Antimicrobial therapies for the different infections encountered include Staphylococcal sp (fluoroquinolones, eg. Exocin); gram-negative rods (pseudomonas); and Chlamydia (tetracyclines).

Other therapies include anti-viral therapies: famciclovir, ganciclovir, interferon and lysine; and anti-collagenase therapies, eg, serum.

Serum is a popular anti-collagenase. Collagenase is produced by some bacteria especially gram negatives, such as pseudomonas. Collect the serum sample, allow to clot, remove the serum fraction, put into an eye-drop container, store in the fridge and replaced every few days. Can you use frozen serum, and can be from other species.

One should have a clear protocol for re-examination of ulcers, which can be extended in the case of superficial ulcers to more like seven days.

New options for treatment of corneal ulceration includes the use of iontophoresis and corneal-cross linkage.

IONTOPHORESIS

Essentially, by creating opposite electrical charges between the natural charge of the molecule (an antibiotic) and the receiving tissue, the cornea, the penetration of the molecule into the tissue is greatly enhanced. It is an option for treating corneal infections.

If riboflavin is used, then another option for treatment of corneal disease is corneal-cross linkage.

CORNEAL-CROSS LINKAGE

Riboflavin is impregnated deep into the corneal stroma by the use of iontophoresis. The cornea is then irradiated with ultraviolet A light. This induces the corneal stiffening and also induces resistance to collagenase digestion. It is a new therapy for corneal ulceration.

SURGICAL OPTIONS

Surgical treatment includes:

- Debride and scarify;
- Conjunctival graft;
- Corneoconjunctival graft;
- Pig-bladder membrane graft; and
- Amniotic membrane graft.

DIFFERENT APPROACHES TO THE CORNEAL ULCER APPROACH TO THE 30% DEPTH CORNEAL ULCER

- Try and decide re the cause of the ulcer. Assess what role facial anatomy may play in the healing of the ulcer and consider infection – culture and sensitivity;
- Medicate atropine drops, uveitis, sid three to four days;
- Broad-spectrum topical antibiosis, considering likelihood of *pseudomonas* Ofloxacin (Exocin) gid;
- Corneal lubrication, to promote healing and for comfort
 OptixcareEye Plus tid;
- Review every 48 hours, neuromuscular blocker may require a surgical approach.

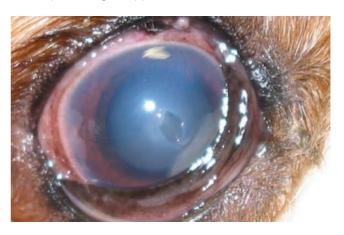


Figure 17: Corneal ulcer with 30% depth.

APPROACH TO THE 30-98% DEPTH CORNEAL ULCER

- Again, assess the likely cause, often a corneal trauma;
- Assess any factors that may complicate healing;
- Assess the likelihood of achieving healing by a medical versus a surgical approach; and
- If deciding on a surgical approach consider what methods you have in your arsenal.

It is important in this analysis to consider that you have the correct microsurgical instruments and the correct suture materials -usually performed with 8/0 –9/0 PGA with a micropoint-spatulated needle. A magnifying loupe of good optical quality is a big advantage.

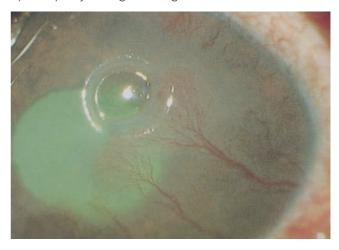


Figure 18: Descemetocoele - 95%.