Conjunctival surgery in the dog

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The conjunctiva is a thin, translucent mucous membrane overlying the anterior portion of the sclera and the inner surface of the eyelids. It is divided into the bulbar conjunctiva, the palpebral conjunctiva, and the superior and inferior fornices. Part of the bulbar conjunctiva covers the internal part of the third eyelid and, part of the palpebral conjunctiva covers the external one, being both of them recognized as the nictitans conjunctiva. It is composed of a non-keratinized, stratified, squamous epithelium and substanția propia. The conjunctiva plays an important role in tear dynamics, ocular movements, immunologic protection of the eye, and corneal healing.

The vascular supply of the conjunctiva is very extensive, allowing adequate and fast healing, but, at the same time, rapid and intense inflammation (i.e. chemosis, hyperemia, blepharospasm, and cellular exudation).

Both, the substancia propia of the palpebral conjunctiva and the bulbar conjunctiva near the limbus, are tightly adhered to the tarsus and Tenon’s capsule, respectively. The majority of the bulbar conjunctiva is freely movable, allowing ocular movements and facilitating some procedures such as biopsies under topical anesthesia, subconjunctival drug injections and some surgical procedures.

In human ophthalmology, conjunctival surgery has changed significantly over the last two centuries. At the beginning of the 20th century, conjunctival surgery was mainly used for the treatment of primary conjunctival conditions, as well as for fixing corneal transplants before direct corneal sutures were precluded. At the end of the 20th century, in which corneal transparency and visual acuity were highly desirable, conjunctival surgery was mostly reduced to primary conditions or as a first surgical approach of certain cases of severe corneal infections/malacia (melting ulcers). In those corneal cases, once the healing was completed, the inflammation controlled, and the eye in “calm”, a second surgery was commonly performed to achieve corneal transparency. Conversely, in the present century, corneal infectious diseases are commonly treated with different types of “therapeutic corneal grafts. Furthermore, conjunctival surgery is also used to transfer ocular motility/fixation to the ocular prosthesis or odontokeratoprosthesis after enucleation.

In veterinary ophthalmology, conjunctival surgery is more frequently performed, mainly due to the tissue availability, its vascular and “nutritional” supply, and its fast healing rate. Simply put, conjunctival surgeries can be classified into primary conjunctival surgeries -if conjunctiva is the diseased tissue-, or secondary conjunctival surgeries -if other tissue is the affected, such as cornea, sclera or eyelids.

Primary conjunctival surgeries include:
- Foreign bodies extraction
- Conjunctival lacerations repair
- Tumor resection
- Conjunctival proliferations surgeries (nonneoplastic masses)
- Symblepharon

Secondary conjunctival surgeries can be further subdivided, based on its purpose, into;
- Protection and healing:
  - Melting corneal ulcers: offer vascular supply and healing factors.
  - ...
● Filling spaces:
  ○ Stromal ulcer/descemetocele: to be used alone or together with other tectonic support, such as corneal grafting, porcine small intestinal submucosa…
  ○ After superficial or deep keratectomy: corneal abscess, corneal or scleral tumors, feline corneal sequestrum,…
  ○ Corneal or scleral thinning
  ○ ...

● Absorption:
  ○ “Letterbox technique”: for the treatment of corneal endothelial disorders (i.e. endothelial degeneration)

In veterinary medicine, many conjunctival grafts have been described, including island, pedicle (bulbar and tarsal conjunctival), bridge, advancement (hood and 180 degrees), and complete bulbar (360 degrees) grafts. The selection of the surgical technique depends on different factors, such as: size, location, depth, presence of infection, instrumentation available (including surgical microscope) and, last but not least, the surgeon ability and experience. Furthermore, other important factors such as advantages, disadvantages and limitations of the chosen technique, need to be considered. The main disadvantages of conjunctival corneal grafting include poor watertightness (sealing), poor tectonic support if used alone, and important postsurgical opacity.

The mainstays of conjunctival corneal grafting are nontraumatic tissue manipulation and advanced knowledge of specialized ophthalmic instruments. The main aspects of the tissue manipulation are summarized below.

● Nontraumatic handling of the conjunctiva (avoid to grape the conjunctiva at many different places).
● Conjunctival graft thickness required (based on the disease or the chosen surgical technique).
● Preservation of blood supply.
● Preparation of the “graft bed”.
● Correct suture technique (including surgical skills, size, needle and suture material selection).

Postoperative treatment depends on the initial corneal disease, but as systemic medication will reach the surgery site by the conjunctival vessels, topical medication rate is commonly diminished. Generally, it is recommended to sever the pedicle base from the limbus at 5-8 weeks postoperatively, to cut off its blood supply, and thus reduce the corneal opacification. Nevertheless, some ophthalmologists, based on some complications described after graft cutting, prefer to do so much later (3-6 months) or not to cut it.

While the anatomical and visual success of primary conjunctival surgeries is high, there are some controversies in relation to secondary conjunctival surgeries’ outcome. The anatomical outcome of conjunctival corneal grafting is generally very high, although visual outcomes are diverse, based on the surgical technique, the location and the size of the graft. Nevertheless, some complications - related to the triggering condition or to the surgical technique- can decrease the anatomical outcome. Most frequent complications include suture dehiscence, flap retraction, thinning, melting/necrosis, tearing and lacerations and impossibility of complete eyelid closure -due to the thickness of the conjunctival flap (“entrecôte mass effect”).

Suggested references


**Lens diseases and its management in the dog**

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**Anatomic and metabolic review**

The lens is a transparent, avascular, biconvex body with an anterior surface that is flatter or less curved than the posterior surface. Briefly, the lens consists of the capsule, anterior epithelium and lens fibers. It is divided into two general regions, the cortex (outer areas near the capsule) and the nucleus (central area). As the lens grows through life, layers of fibers are produced in the equatorial area and are laid down on top of the former layers, forcing older fibers towards the lens center in a process resembling the formation or rings in tree trunks. The lens is supported at the equator by the lens zonules, or suspensory ligaments. Alterations of tension in these fibers may induce lens movement, and in severe cases, lens displacement.

Because the lens is avascular, its metabolic needs are met by the aqueous humor. Therefore, lens metabolism is precarious and depends on constant composition of the aqueous. Disturbances in aqueous composition affect lens metabolism and transparency.

**Lens diseases classification**

Lens diseases in small animals can be classified as congenital or acquired. Congenital lens diseases, although rare, are worth mentioning, being the most important aphakia, microphakia, spherophakia, lenticonus, coloboma and cataracts. Acquired lens diseases are much common and easily detected by the owners, due to their evident clinical signs. The most frequent include: cataracts, lens luxation or subluxation and lens sclerosis.

**Cataracts**

The term cataract comprises a common group of ocular disorders manifested as loss of transparency of the lens or its capsule. The opacities may be of varying sizes, shapes, location within the lens, etiology, age of onset, and rate of progression. Because of the variable nature and appearance of cataracts, numerous methods of classification are commonly used, such as:

- **Stage of development**: incipient, immature, mature, hypermature, Morgagnian
- **Position within the lens**: anterior capsular, anterior subcapsular, cortical, equatorial, nuclear, posterior subcapsular, posterior capsular
- **Age of development**: congenital, developmental, juvenile, senile, acquired
- **Etiology or pathogenesis**: primary (inherited) or secondary (traumatic, intraocular disease, nutritional, metabolic, radiation, toxic, senile, congenital abnormalities.
- **Consistency**: fluid, soft or hard.

Lens opacification if mild, or located in the periphery of the lens (not affecting the visual axis), can be difficult to detect by the owners, as no evident clinical signs are commonly seen. The most common cataracts seen in the dog are hereditary, thus the importance of detecting the affected animals and setting them apart from breeding. A big deal is that many of the hereditary cataracts are phenotypically seen in adulthood, when the animals already have progeny. There are some genetic tests available at the moment, but unfortunately not for all the breeds and not for all the affected genes. Thus, if the genetic test is positive the cataract has a hereditary basis, but if negative, this cannot be ruled out.
The gold treatment for the canine cataracts nowadays is phacoemulsification and intraocular lens implantation (+41D). The surgery should be recommended when cataracts induce a visual deficit. A complete blood work, electroretinography, ocular ultrasound and gonioscopy should be performed as presurgical workout. The results of these complementary tests would modify help to select the most appropriate treatment for each candidate. The selection of the candidates for phacoemulsification is very important, increasing in that way, the success (visual eye) of the surgery (98% in our center). The premedication varies depending on the surgeon and the patient, being the most frequent topical antibiotics, topical NSAIDs, topical corticosteroids, topical phenylephrine, topical tropicamide, and systemic antibiotics and NAIDs. Different techniques of phacoemulsification have been described and will be discussed during the lecture. Post-operative treatment and management of the patients is extremely important as well, and varies depending on the surgeon’s preferences, the surgery, the patient and the owner. Different options will be discussed during the lecture.

**Lens sclerosis**

Lens sclerosis, or “greyish lens”, is part of the aging process and occurs in both, animals and humans. These lesions consist on opacification of the nucleus and appear from 6-7 years old. Its progression is low, and in pet dogs is not associated with vision deficits, as the lens accommodation capacity of the dog is virtually absent. The difference between senile sclerosis and senile cataract can be done by means of indirect ophthalmoscopy or indirect retroillumination; lens sclerosis allows posterior segment visualization, while cataracts do not. Lens sclerosis does not need treatment, although an annual recheck is recommended in order to diagnose incipient senile cataracts that do affect vision.

**Lens subluxation and luxation**

Lens subluxation occurs when some of the zonules are torn, leading to displacement of the lens from its normal position, inducing in some cases movement of the lens (phacodonesis). Similarly, lens luxation occurs when all the lens zonules are broken and the lens moves anteriorly or posteriorly (allowing an aphakic crescent to be seen). Normally lens luxation is preceded by lens subluxation, resulting form tearing of some of the zonules, but in many cases its goes unnoticed.

Lens luxation can be primary or secondary. Primary lens luxation is considered an hereditary condition in some breeds, such as the Cairn terrier, Shar-pei, Jack Russell Terrier, Tibetan Terrier, Staffordshire bull terrier,... and appears in adulthood. Normally is bilateral, although not synchronous, nor symmetric.

Secondary lens luxation can appear in cases of glaucoma, intraocular tumors, chronic uveitis, ocular trauma, chronic cataracts.. Commonly it is unilateral and appears in association to other clinical signs such as uveitis, cataract, buphthalmia, hyphema, etc...

The differentiation between primary and secondary lens luxation is performed based on the ophthalmic examination findings and the breed of the affected animal.

The treatment differs significantly if the lens luxation is primary or secondary. In secondary cases, the primary cause needs to be treated first, and when fixed the lens luxation be approached. In primary cases, the treatment of choice will vary depending on different aspects, such as the age of the animal, if the eye is visual or not, if the lens is anteriorly/posteriorly luxated, if there are other intraocular complications (ocular hypertension, synechiae, partial retinal detachment, vitreal syneresis, etc...). The goal treatment for lens luxation is intracapsular lens extraction/bimanual phacoemulsification and, if possible, sulcus lens implantation. The implantation of the intraocular lens can be performed by different techniques that will be briefly discussed during the lecture.
In summary, lens diseases in dogs can be easily diagnosed and successfully surgically treated by an experienced ophthalmologist. The diagnosis in early stages of the disease is imperative for vision maintenance.

**Suggested references**


Management of sudden blindness in the dog

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Sudden blindness is a frequent cause for an ophthalmologic consult in our canine patients. Undoubtedly, it is considered a veterinary emergency, bearing in mind it can lead to permanent blindness if not properly treated right away.

**Differential diagnosis**

Predominantly, sudden blindness can be caused by four main mechanisms: **opacification of the ocular media**, **impaired retinal function**, **impaired function of the optic nerve** and **abnormal function of central nervous system (central blindness)** (Table 1). The most frequently diagnosed mechanism in cases of sudden blindness that is not associated with ocular pain is, undoubtedly, impaired retinal function.

**Table 1. Differential diagnosis of sudden blindness in the dog**

<table>
<thead>
<tr>
<th>OPACIFICATION OF THE OCULAR MEDIA</th>
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<tbody>
<tr>
<td>Anterior uveitis</td>
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<tr>
<td>Cataract</td>
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<tr>
<td>Vitreal hemorrhage</td>
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<tr>
<td>Lens luxation</td>
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<td>Glaucoma</td>
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<tr>
<th>IMPAIRED RETINAL FUNCTION</th>
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<tr>
<td>Retinal detachment</td>
</tr>
<tr>
<td>Corioretinitis</td>
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<td>Retinal hemorrhage</td>
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<tr>
<td>Sudden acquired retinal degeneration syndrome <strong>(SARDS)</strong></td>
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<tr>
<td>Cancer associated retinopathy <strong>(CAR)</strong></td>
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<tr>
<td>Immunomediated retinitis <strong>(IMR)</strong></td>
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</tbody>
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<tr>
<th>IMPAIRED OPTIC NERVE FUNCTION</th>
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<tbody>
<tr>
<td>Optic neuritis</td>
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<tr>
<td>Retrobulbar optic neuritis</td>
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<td>Optic nerve neoplasia</td>
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<table>
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<tr>
<th>IMPAIRED SNC FUNCTION</th>
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<tbody>
<tr>
<td>Meningitis</td>
</tr>
<tr>
<td>Meningoencephalitis</td>
</tr>
<tr>
<td>Neoplasia in different locations</td>
</tr>
<tr>
<td>Vascular abnormalities in different locations</td>
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<tr>
<td>Intracranial pressure increase</td>
</tr>
</tbody>
</table>

**Diagnostic approach**

The initial diagnostic approach is identical to any other ophthalmologic emergency. It starts by complete ocular examination. In cases of sudden blindness, neuro-ophthalmology plays a crucial role (visual placing
response, dazzle reflex, papillary light reflexes, corneal reflex and palpebral reflex), specific vision tests must be performed (maze test) in both photopic and scotopic conditions (light and dim conditions respectively) and bilateral fundus examination is required.

The part of the ophthalmologic examination that generates many doubts for the general practitioner is definitely the fundus examination. Given the wide variety of physiological fundus, particularly in dogs, minimal experience is essential to detect, distinguish and diagnose eye fundus abnormalities. This task can be facilitated by the combined use of direct and indirect ophthalmoscopes, allowing in this way a complete and detailed assessment of the ocular fundus.

The complete ocular examination can detect many intraocular problems, such as severe uveitis, cataract, glaucoma, retinal detachment, optic neuritis, etc ... (Fig.1). In these cases, it is recommended to build up specific etiologic differential diagnosis, to extend the diagnostic protocol, and to establish a visual prognosis and initiate specific medication for the problem that has been diagnosed.

In case that the ophthalmological examination does not reveal any important intraocular changes, it will be temporarily considered as “amaurosis” (blindness of unknown origin) and further diagnostic trials are required in order to reach a definitive diagnosis. One of the main procedures at that instance is the electroretinography (ERG) (Fig.1), the short protocol of assessment of retinal function being the most used (the so called ON/OFF protocol). If ERG demonstrates adequate retinal activity, the diagnostic possibilities are restricted to retrobulbar optic neuritis or central blindness. In this case it is indicated to perform complete neurological examination, MRI and cerebrospinal fluid analysis. If, however, the ERG confirms the absence of retinal activity, the differential diagnosis includes diseases such as sudden acquired retinal degeneration syndrome (SARDS), cancer-associated retinopathy (CAR) or immunomediated retinitis (IMR). In those cases, and in order to reach an etiologic diagnosis, the diagnostic protocol must be extended including colorimetric pupillary reflex test, thoracic radiographic studies, abdominal ultrasound, blood count and biochemical tests, urinalysis, sex hormones plasma levels (17-hydroxyprogesterone, estradiol), ACTH stimulation test, and in some cases, fluorescein angiography and optical coherence tomography (OCT).

![Figure 1. Diagnostic tree for sudden blindness in dogs](image-url)
Most frequent clinical diagnoses

Undoubtedly, the most frequent diagnoses of sudden blindness that are not associated with ocular pain and or with any significant fundus alterations are: SARDS, CAR and IMR.

Sudden acquired retinal degeneration syndrome (SARDS)

Sudden acquired retinal degeneration syndrome or SARDS, is a retinal disorder described in the canine species that causes acute blindness without apparent fundus changes. The most common clinical presentation is sudden blindness associated with non responsive dilated pupils as the only detectable ocular abnormality. Although there are numerous scientific studies that have associated this syndrome to metabolic problems (hyperadrenocorticism) or to punctual or permanent sex hormone increases, currently its etiology remains unknown, enclosing mainly immune-mediated causes.

Cancer-associated retinopathy (CAR)

CAR or cancer-associated retinopathy is a retinal disorder, similar to SARDS, where sudden blindness occurs without any obvious fundus changes. It is a paraneoplastic immune mediated syndrome, which generates antibodies that cross-react with antigens localized in the retina. This immune response directed against the retina leads to retinal cell death and induces acute visual loss. Despite being a pathology well documented in human medicine, few cases have been described in veterinary literature, mainly associated with neoplasms such as melanomas and carcinomas. In most of these cases, blindness appears before diagnosis of the tumor, being irreversible.

Immune-mediated retinitis (IMR)

Retinitis or immune-mediated IMR is a retinal disorder, similar to the above, that occurs with no apparent fundus changes. The specific pathogenesis of this disease, despite being immune-mediated, has not been established. Together with SARDS, it encloses all cases of sudden retinal blindness whose cause is not associated with systemic abnormalities.

Visual prognosis

The visual prognosis varies depending on the cause that generated blindness and on the chronicity of the process. In the case of SARDS, CAR and IMR, visual recovery prognosis is reserved, even though there have been some reports of partial visual recovery.

Treatment

Treatment of sudden blindness depends on the cause and, consequently, it may be medical or surgical. Regardless of the type of treatment required, it needs to be initiated as soon as possible in order to avoid irreversible visual damage. The treatments described for the three most frequently diagnosed pathologies are summarized below, some of them still being in experimental clinical phase.

In the specific case of SARDS, although until recently considered an incurable disease, studies advocate treatment with a combination of human immunoglobulin (IVIg) intravenously. The University of Iowa described the treatment protocol in 2008. This treatment is recommended in early cases when evident retinal atrophy is not present. The main problem is that being of human origin, the intravenous administration implies a high risk of anaphylactic reaction. The effectiveness of this treatment is not clearly defined, currently being still in clinical testing phase.
In cases of diagnosed CAR, treatment protocol is based on the specific treatment of the tumor and may or may not include adjuvant immunosuppressive therapy. In most cases the chemotherapy protocol is enough to induce immunosuppression, while in others it must be combined with prednisone (1-2 mg/kg BID) and doxycycline (10 mg/kg BID).

Referring to cases of IMR, there are different documented protocols, being the combination of prednisone (1-2 mg/kg BID) and doxycycline (10 mg/kg BID) the most used one. However, the percentage of visual recovery remains low.

Conclusions

Early diagnosis and proper immediate treatment are the keys to preserve vision in cases of blindness from retinal origin, in the absence of ocular pain, and with no pathologic fundus findings. Still, the prognosis for visual recovery in these cases remains guarded to poor.

Suggested references


Clinical approach to the red eye in the dog

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The “red eye” is one of the most common clinical presentations in veterinary ophthalmology accounting, together with blepharospasm, for approximately 75% of all referral cases. It is a common and nonspecific clinical sign that may accompany multiple eye disorders; therefore, it is important to point out that it is not always a synonym for conjunctivitis. Eye congestion often occurs simultaneously with other ocular signs (specific and nonspecific), thus establishing the ophthalmologic clinical presentation.

Only after a complete ophthalmologic examination, the veterinarian can issue a definitive diagnosis, with conjunctivitis being one of the possible differential diagnoses. This lecture provides the necessary tools to deal with the diagnostic approach of these cases with greater safety and efficiency.

Anatomical and physiological review

Anatomy

Eye hyperemia, colloquially known as “red eye”, may affect the conjunctiva that covers the eyelids’ inside (palpebral conjunctiva), the conjunctiva found around the globe and anchored in the esclerocorneal limbus (bulbar conjunctiva), and also the sclera or the episclera (deeper vessels).

The conjunctiva is formed by:

- Non-keratinized stratified squamous epithelium, which contains goblet cells that are responsible for the secretion of the mucoprotein layer of the tears film.
- Substantia propia, where one can localize the lymph nodules (surface layers), and the vessels and nerves (deeper layers).
- Both the bulbar and palpebral conjunctivas are highly vascularized structures and, depending on the dog’s breed, they may present different degrees of pigmentation. The palpebral conjunctiva is defined as thick, opaque, pink and motionless; on the other side, the bulbar conjunctiva is thinner, semitransparent and mobile.

Physiology

The conjunctiva is involved in the tear film dynamics; it protects the eyeball, facilitates its movement and is often necessary for the process of corneal scarring. Being one of the easiest ocular structures to examine directly, alert owners can detect small changes in the early stages of the process. Due to its rich vascularization, the conjunctiva usually responds to aggression by marked vasodilatation, leading to the characteristic appearance of “red eye”. In addition, the fact that the bulbar conjunctiva is thin and transparent allows the observation of deeper vessels located in the different scleral layers. These vessels, which under normal conditions are almost imperceptible, in cases of corneal or intraocular pathology can expand and increase in number, giving an appearance of “red eye”.

In conclusion, eye hyperemia can be either palpebral (when the palpebral conjunctiva is affected) or bulbar. In cases of bulbar congestion it is important to differentiate between superficial vessels (bulbar conjunctiva) and deep vessels (sclera and episclera). This differentiation can be performed by several methods, including:
● Experienced ophthalmologist, due to the vessel appearance and thickness.
● Mobility with a sterile swab, bearing in mind that the deepest structures present less mobility compared to the more superficial ones.
● Vasoconstriction induced by a sympathomimetic agent, such as phenylephrine. An application of one drop of phenylephrine will lead to the absence of conjunctival congestion, but scleral congestion will remain...

**Differential diagnosis of red eye**

The clinical sign of “red eye” may occur in multiple ophthalmologic diseases. Hyperemia is often secondary to inflammation of the ocular surface, but occasionally it may be associated with an intraocular alteration, being then a symptom of a more severe disease.

Fortunately, many patients with red eye have only mild ocular inflammation, which may be due to different pathologies, such as **conjunctivitis and dry eye syndrome or keratoconjunctivitis sicca**. In other cases, conjunctival congestion may be associated with more severe corneal or intraocular disorders, including **corneal ulcers** of different depth and severity, **non-ulcerative keratitis** (pigmentary pannus, eosinophilic keratitis, stromal keratitis, corneal abscess, etc.), **scleritis or episcleritis, anterior uveitis, glaucoma**, and **lens luxation**, among others.

When eye congestion is limited to the palpebral conjunctiva it is principally associated with conjunctival processes, meanwhile bulbar congestion can go further associated with corneal and intraocular processes. It is vital for the clinicians to diagnose what is causing conjunctival hyperemia in the eye in the early stages of the disease in order to start appropriate treatment as soon as possible and to avoid sequelae. Each disease has its own specific treatment, which can be counterproductive to other diseases, so the correct ophthalmologic diagnosis holds the key to therapeutic success.

**Red eye diagnostic approach**

The correct approach to ocular congestion begins with a complete and systematic ocular examination. Some of the exploratory phases can be of great diagnostic value, showing pathognomonic signs of very specific alterations, such as the following eye diseases: reduced Schirmer Tear Test value (KCS), aqueous flare (uveitis), aphakic crescent (subluxation or luxation of the lens), increased intraocular pressure (glaucoma), fluorescein uptake of the corneal surface (corneal ulcer), etc.

Below you will find some flow charts that can be helpful in addressing the “red eye” in the dog. In addition, the most frequently observed diagnoses in daily clinical practice are listed.
FLOW CHART OF THE "RED EYE" IN THE DOG

**CONJUNCTIVITIS**
- Palpebral
- Bulbar

**HIFEMA, FIBRINA, HIPÓPIA, PKS, RUBEOSIS IRIDIS, EDEMA IRIS,...**

**DIAGNOSES**
- KCS
- < 15 mm
- > 15 mm

**1. STT1***
- Menace response: non-specific
- Dazzle reflex: non-specific
- Pupillary light reflexes: non-specific
  - Miosis: may suggest uveitis
  - Mydriasis: may suggest glaucoma

**2. NEUROPHTHALMOLOGY**

**3. CONJUNCTIVAL HYPEREMIA**

**4. ADNEXA EXAMINATION**

**5. CORNEAL/SCLERAL EXAMINATION**

**6. ANTERIOR SEGMENT EXAMINATION**

**7. AQUEOUS FLARE***
- Positive
- Negative

**8. TONOMETRY***
- > 20-25 mm Hg
- < 20-25 mm Hg

**9. FUNDUSCOPY**

**10. FLUORESCEINE***
- Positive
- Negative

**DIAGNOSES**
- LENS LUXATION/SUBLUXATION
- UVEITIS
- GLAUCOMA
- CORNEAL ULCER

**SPECIAL CONSIDERATIONS**
- More indicative of corneal/intraocular disease

**FLOW CHART OF THE "RED EYE" IN THE DOG**

*Pruebas de gran valor diagnóstico en el diagnóstico de ojo rojo de aparición aguda*
CLINICAL APPROACH TO GLAUCOMA IN THE DOG

↑IOP

Miosis +/- signs of uveitis (Tyndall, hifema, ...)

Mydriasis

Gonioscopy

Secondary glaucoma
 Hypertensive uveitis

Open angle (rare)

Primary open angle glaucoma POAG

Ocular fundus examination

Few retinal findings

Uveitis diagnostic protocol

Medical treatment & surgical treatment

Visual eye ?

Mydriasis

Gonioscopy

Closed/narrowed angle or PLD

Primary open angle glaucoma POAG

Ocular fundus examination

Few retinal findings

Medical treatment & surgical treatment

Repeat tonometry

Chronic glaucoma

Surgical & Medical emergency treatment

Differential Diagnosis

Contralateral gonioscopy and differential diagnosis

Anterior lens luxation

Chronic glaucoma

Corneal edema

Buphthalmia normal size

Lens luxation

Buphthalmia

Ocular ultrasound

Signs of inflammation or tumor

No signs of inflammation

Primary glaucoma

Secondary glaucoma

Buphthalmic eye

Non visual eye

Gonioscopy contralateral eye

Glucoma closed, narrow or PLD

Normal size

Buphthalmic eye

ONH atrophy

Few retinal findings

Glaucoma
Canine and feline corneal ulceration medical versus surgical treatment

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Cornea is made up of the epithelium, stroma and endothelium. The main function of the corneal epithelium is to act a physical barrier against pathogens. The corneal epithelial cells are highly responsive to injury. Epithelium is a lipophilic barrier, which means it will repel water and maintain the rest of the cornea at a dehydrated state. The corneal epithelium is made up of 2 to 3 layers of squamous cells, which they are non-keratinized, have microvilli on their surface which has been proposed to help anchor the mucin part of the tears onto the cornea. Below the squamous cells are the “Wing” cells and the basal epithelial cells which is columnar in shape, have desmosomes with adjacent cells and hemidesmosomes with anchoring fibril to the basal lamina.

The corneal stroma especially the anterior portion and the basal lamina of the corneal epithelium are important structures for corneal wound. The anterior stroma has very few keratocytes compared to the rest of the stroma but it has type IV collagen, laminin and fibronectin and these collagen fibrils anchors onto the basal lamina of the epithelium. The posterior portion of the stroma has high concentration of keratin sulfate, which it can absorb water 2 to 3 times higher than chondroitin sulfates. The later however can retain water 8 to 9 times as much as keratin sulfate. At the anterior stroma there is a much higher concentration of dermatan sulfate.

The last layer is the endothelium and its basement membrane, the Descemet’s membrane. This membrane has some elastic properties. The endothelium is a monolayer of polygonal cells. In most adult animals these cells are nonregenerative. Therefore, when damaged, these cells will not be replaced. However, the adjacent cells can undergo metaplasia by hypertrophy to cover the defective area and continue to maintain the integrity of the endothelium. The number density for endothelial cells is 3000 cells/mm$^2$. Corneal endothelial cells will undergo hypertrophy when the cell density is less than 800 cells/mm$^2$. Corneal edema will occur when the cell density is less than 500 cells/mm$^2$.

There are certain dystrophic and degenerative corneal diseases that initially does not lead to ulceration but over time corneal ulceration may occur.

Dystrophies and degeneration

Corneal epithelial dystrophies, corneal stromal dystrophies and degeneration, corneal endothelial dystrophy and feline bullous keratopathy are a group of diseases that affect the different layers of the cornea causes cornea to have either focal or diffuse opacity, keratitis or edema. In the early stages of the diseases, no corneal ulceration is noticed, however with protracted time, corneal ulceration can be associated with these condition and medical therapy will be required. If the ulcer is deepened due to infection, surgical intervention will be required.

Immune mediated corneal diseases

These include condition such as chronic superficial keratitis in dogs and eosinophilic keratitis in cats. Infiltration of lymphoplasmacytic cells or eosinophils are noticed and corneal ulceration may or may not be present. Typically, topical steroid and/ or cyclosporine should control these conditions.
Feline sequestration

Feline sequestration is a poorly understood condition in terms of its etiology and pathogenesis. Management of this condition depends on the dedication of the owner and if the condition has caused serious morbidity to the cat. Both medical and surgical intervention can be considered and depends on the depth of the sequestration.

Spontaneous chronic corneal epithelial defect (SCCED)

Spontaneous chronic corneal epithelial defect or indolent corneal ulceration is another corneal ulcerative condition that we do not fully understand its etiology and pathogenesis. Altered corneal innervation, trauma, irritation (such as altered tear distribution or quality), dysplasia of basal epithelial cells has been proposed or an unknown organism - in the case of cats, sometime herpes viral infection can result in SCCED. This condition can be treated with cotton bud debridement, grid keratotomy, superficial keratectomy or diamond burr keratectomy. The healing rate and success of each procedure will differ.

Stromal corneal ulceration

The choice of antibiotic will depend on the type of ulceration and also if necessary base on culture and sensitivity and cytology collected. In simple none infected epithelial ulceration, a broad spectrum bacteriostatic antibiotic should be sufficient. We have to understand that the antibiotics itself and also the preservative within can inhibit epithelisation or cause toxicity to the keratocytes. If cornea is presented with deeper ulceration, apart from ensuring the appropriate antibiotic is chosen, we need to be sure that the antibiotics has good penetration and least toxic to the keratocytes.

Lastly, the underlying cause of the ulceration should also be treated. Such as if the corneal ulceration is due to keratoconjunctivitis sicca, Optimmune or Cyclosporine and artificial tears should be added into the treatment regime. Entropion or ectopic cilia are also common causes of corneal ulceration, therefore these adnexal condition should be corrected or removed respectively. Conjunctival pedicle graft, corneoscleral transposition graft, Vet BioSiST®/ ACell VetTM graft or amniotic membrane graft should be considered in those stromal ulcers that are more than ½ stroma in depth.

Keratomalacia

Keratomalacia is an extension of any kind of corneal ulceration where the proteolytic activity either from the keratocytes, neutrophils, bacteria (such as pseudomonas aeruginosa), fungal (such as aspergillus) or chemical in particular alkali burn that is not counteracted or the proteases are not inhibited. Serinase protease, matrix metalloproteinases 2 and 9 are some of the more active and significant proteases that can devastate a cornea.

Keratomalacia can be managed by either medically or surgically. The decision to proceed with medical or surgical therapy depends on the depth of the keratomalacia and the health of the patient. Successful outcome has been achieved with both medical and surgical therapy. Corneal culture and sensitivity and also corneal cytology should be performed in order to select the most appropriate topical antibiotic.

Surgical therapy is generally considered if the keratomalasic condition is protracted, with no healing response, deteriorating or perforated. Conjunctival pedicle graft, corneoscleral transposition graft, SIS graft, third eyelid transposition graft, 360-degree conjunctival graft, amniotic membrane graft has all been described. The success rate for resolution is very high with surgical intervention.
References


Canine keratoconjunctivitis sicca (KCS) - Possibility of dry eye treatment with diquafosol natrium -

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On the surface of the eye, the tear film consists of several layers: a mucin layer covers the ocular surface, an aqueous layer lies above the mucin layer, and a lipid layer covers the tear film surface. Quantitative tear deficiency refers to a lack of the aqueous layer. Qualitative tear deficiency refers to a lack of the mucin and/or lipid layer.

Quantitative and/or qualitative tear deficiency in dogs is called canine keratoconjunctivitis sicca (KCS). The most common etiology is immune-mediated lacrimal gland adenitis in the dog.

The therapeutic strategy for immune-mediated KCS is typically focused on improving the quantitative tear secretion. In the initial treatment instance, the administration of a temporary artificial tear formulation, for example, a cyclosporine A ointment, can alleviate the severe lack of tears associated with KCS. On the other hand, qualitative tear deficiency commonly occurs in brachycephalic and/or lagophthalmic dogs. In these cases, a typical therapeutic approach might be the topical application of hyaluronic acid.

Recently, a new ophthalmic medication, diquafosol natrium, has been approved in Japan for the treatment of dry eye in humans. Diquafosol is a P2Y2 purinergic receptor agonist. In a rabbit model, diquafosol has been shown to act on the P2Y2 receptors to stimulate water secretion from the conjunctival epithelial cells and mucin secretion from the conjunctival goblet cells, and has been shown to improve the corneal epithelial integrity in experimental dry eye disease. Furthermore, the use of diquafosol eye drops in human KCS patients led to tear film stabilization and subsequent improvement in the tear break-up time, as well as improved fluorescein and rose bengal vital ocular surface staining scores. The expression of the P2Y2 receptor has not previously been evaluated in the canine conjunctiva. If the receptor is present, it is feasible that use of diquafosol would increase the aqueous and mucin secretion, which might help to stabilize the tear film in canine ocular surface disorders. This would be reflected in an increase in the tear break-up time, which could lead to improvements in the treatment of corneal epithelial disorders, such as superficial punctate keratopathy. The aim of the present study was to examine the expression of canine conjunctival P2Y2 receptors and assess the effect of topical diquafosol on the ocular aqueous and mucin secretion.

The canine conjunctival P2Y2 receptor expression was evaluated by Western blotting and immunohistochemical analysis. The effect of diquafosol on mucin secretion was evaluated by measuring the concentration of mucin-5 subtype AC (MUC5AC) in the tears. The effect of diquafosol on the aqueous secretions was evaluated by performing the Schirmer tear test (STT) and phenol red thread test. The expression of the P2Y2 receptor was confirmed in the canine bulbar and palpebral conjunctivae, and the receptors were identified at the conjunctival epithelial and goblet cell surface. The tear MUC5AC concentration was significantly increased after the administration of a 3% diquafosol ophthalmic solution, although neither the STT nor phenol red thread test values showed any significant change after the diquafosol instillation. The topical ocular administration of 3% diquafosol might improve the symptoms of corneal epithelial disorders in dogs through the stabilization of the tear film due to the increase in MUC5AC secretion in the mucin layer. It is useful for the alleviation of the symptoms associated with qualitative tear deficiency in canine KCS.