

UVEITIS IN DOG AND CATS

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Definition

Uveitis can be defined as an inflammatory process of the uveal tract. Clinically, the iris and ciliary body usually react together, while the choroid behaves somewhat independently. Uveitis is thus generally classified into two categories: anterior uveitis or iridocyclitis and posterior uveitis or choroiditis (1).

The uveal tract is a highly vascularized and heavily pigmented structure which constitutes the middle layer of the eyeball, between the outer fibrous tunic (sclera) and the inner nervous lining (retina). The size of the pupil is the result of a balance between the action of the dilator muscle (adrenergic innervation) and that of the sphincter muscle (cholinergic innervation). Clinically, it is particularly important to remember that the iris sphincter is much more potent than the dilator. The ciliary body is the only part of the uveal tract that cannot be visualized during a routine ocular examination: hidden from direct view by the iris, it is located too far anteriorly to be observed with the ophthalmoscope. The ciliary body has many roles, one of the most important is the formation of vitreous humor. Any change in the composition or in the rate of flow of the aqueous humor can have serious deleterious effects.

The choroid is the most posterior portion of the uveal tract. It has an important role for the nutrition of the posterior retinal layers (2).

Etiology of Uveitis

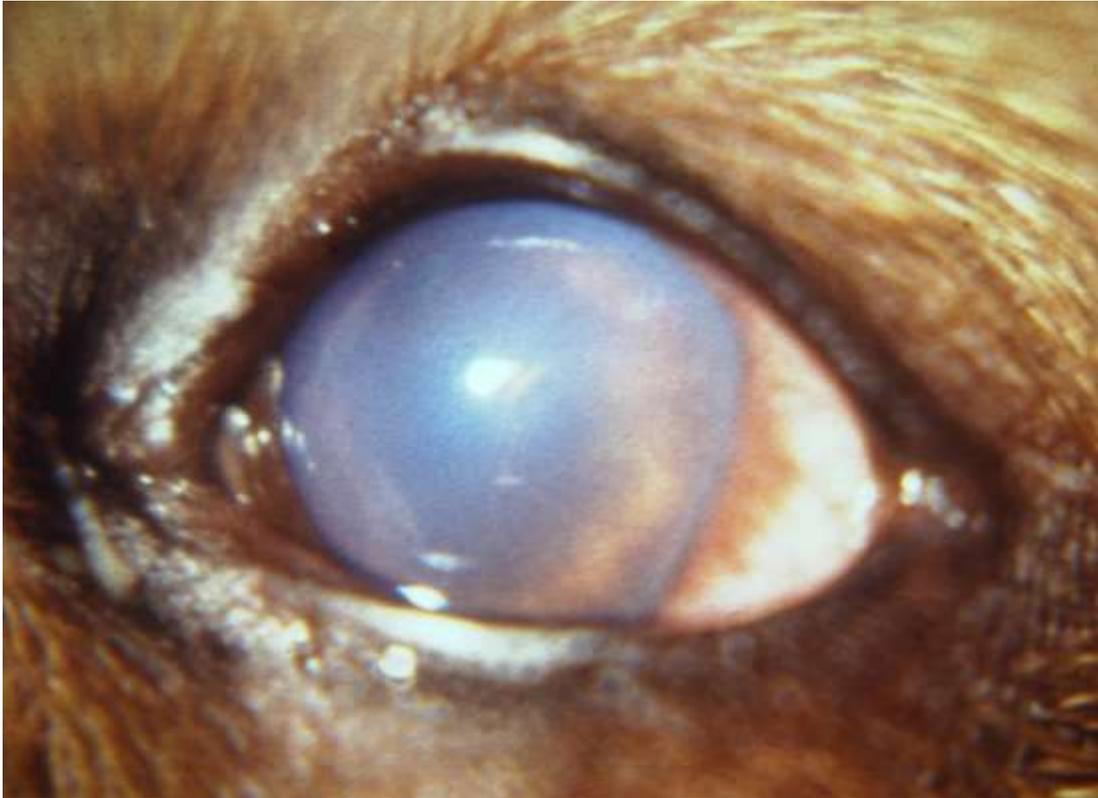
While there are many known causes of uveitis, a specific etiological factor cannot always be determined. It is probable that immune mechanisms are frequently responsible for the development of inflammation. Presently, the term "idopathic" is often used to qualify such cases for lack of a more precise etiology (1).

Trauma is an important cause of iridocyclitis in animals. Whether blunt or perforating, trauma causes the release of intraocular prostaglandins and the classic signs of inflammation. Corneal ulceration is another condition associated with anterior uveitis (3).

A variety of bacteria can cause uveitis. For instance, exogenous infections can occur after perforation of the globe. Notable exceptions include brucellosis, leptospirosis and tuberculosis, diseases in which microorganisms can often be recovered from the eye. In systemic mycoses such as blastomycosis, cyptococcosis, histoplasmosis and coccidioidomycosis, intraocular infection readily occurs, usually in association with a granulomatous inflammatory reaction.

Intraocular parasites are possible causes of uveal inflammation. *Toxoplasma gondii*, the most commonly encountered ocular parasite in dogs and cats, can be associated with both iridocyclitis and retinochoroiditis. *Dirofilaria* has also occasionally been seen in the eye (4, 5).

Viral infections can be the trigger of anterior or posterior uveitis. The iridocyclitis associated with canine adenovirus type I infection (natural or vaccinal) has been extensively studied. Pic. 1.



Pic. 1. Dog with uveitis – after vaccination

The feline leukemia (FeLV) and the feline infectious peritonitis (FIP) viruses are also frequently associated with uveitis. Finally, ocular neoplasms, whether primary or secondary, often induce or mimic an inflammatory reaction (6, 7). Pic. 2.



Pic. 2. Dog with lymphoma causing bilateral uveitis.

Clinical Signs of Uveitis

There are three clinical signs associated with uveitis: pain, decreased vision and a change in the appearance of the eye (1).

Ocular pain, manifested mainly by blepharospasm and photophobia, is the most frequent sign of uveitis. It is seen only with inflammation of the anterior uvea, as the choroid and retina lack afferent nerve fibers. The degree of pain associated with iridocyclitis varies greatly and appears to depend on the type of inflammatory reaction and on its intensity. As a rule, acute anterior uveitis induces much more pain than a chronic, granulomatous type of inflammation. In animals, decreased vision is seldom noted by the owner unless it is bilateral and pronounced. The sudden onset of blindness is a true emergency. Loss of vision is actually the sole clinical sign of posterior uveitis. (1, 2, 8)

Clinical Findings in Anterior Uveitis

General findings

Pain and photophobia are often obvious when the eye is examined. When present, ocular discharge is usually serous, as a result of increased lacrimation. Vascular congestion is a common finding in iridocyclitis. Episcleral vessels are usually dilated (3).

Corneal changes

The cornea may show a diffuse edema. Corneal edema is generally associated with acute, intense inflammation. It is caused by damage to the corneal endothelium. Corneal vascularization is seen with severe chronic or recurrent diseases. Vessels migrate for a short distance from the limbus into the deep corneal stroma over the entire corneal circumference. This type of vascularization has been termed "paint-brush" in appearance, as compared with the arborizing pattern seen when vessel invasion is superficial (2).

The presence of keratic precipitates is always indicative of iridocyclitis. These are inflammatory cells, usually mononuclears, which become plastered against the posterior corneal surface as they are carried by the convection currents of the aqueous humor. They are usually seen in the inferior half of the cornea (4).

The aqueous humor

The aqueous humor is markedly altered in the course of iridocyclitis. If the outflow channels are still patent, this results in a net decrease in intraocular pressure: hypotony is typical of anterior uveitis. The intraocular pressure should always be evaluated in the case of a red, painful eye. Tyndall phenomenon in the aqueous humor - in a normal eye, the protein content of the aqueous humor is extremely low due to the intact blood aqueous barrier. Anterior uveal inflammation destroys this barrier, allowing serum proteins to leak into the aqueous humor. As a result, a beam of light going through the anterior chamber can be seen as it becomes scattered by the abnormally dense aqueous: the impression is that of a ray of sunlight in a dusty room. In a case of iridocyclitis, the beam of light can also be seen as it travels through the anterior chamber: the higher the aqueous protein content, the denser the beam. When the fibrin content of the aqueous humor is very high, a clot may form, filling part of the anterior chamber. White blood cells can also be seen in the abnormal aqueous. In some cases, they adhere to the corneal endothelium forming keratic precipitates, while in others, they settle by gravity to the bottom of the anterior chamber, causing a hypopyon. Lipids are another abnormal component of the aqueous humor in occasional cases of uveitis. They impart a very milky, opalescent appearance to the aqueous and are seen only when there is a breakdown of the blood-aqueous barrier occurring simultaneously with severe lipemia (1, 2).

Changes in the iris and pupil

Changes in the color and texture of the iris are commonly seen with anterior uveal inflammation. During the acute phase, the iris becomes thickened and velvety-looking. With chronic inflammation, there is eventually some atrophy of the iris stroma. The pupil is classically miotic in a case of anterior uveitis due to a spasm of the iris sphincter. The formation of posterior synechiae may fix the pupil partially or completely into an irregular form. This follows the breakdown of the blood-aqueous barrier making the aqueous very sticky and causing the iris to adhere to adjacent structures. The presence of synechiae is always indicative of a previous iritis.

Changes in the lens and vitreous humor

Anterior uveitis can result in the formation of an opaque clot crossing the pupil and adhering to the anterior lens capsule. Cataract formation then occurs. If cyclitis is pronounced, outpouring of exudate into the vitreous humor can occur. The exudate may eventually form opaque membranes. Opacities within the vitreous cavity will then appear to float as the eye moves (1, 2, 3, 5, 7).

Pic. 3.



Pic. 3. Dog with uveitis – lens – induced uveitis.

Clinical Findings in Posterior Uveitis

Clinically, it is difficult to make a clear distinction between choroiditis, retinitis and chorioretinitis. Fundic changes associated with choroidal inflammation are more easily observed in the tapetal fundus. Choroiditis results in the local accumulation of an inflammatory exudate. The first change consists of a patchy dullness in the tapetal area. The accumulation of subretinal exudate results in flat, then bullous retinal detachments (4, 7, 9).

Treatment of Anterior Uveitis

Anti-inflammatory treatment

This the single most important part of treatment and, while nonsteroidal anti-inflammatory agents can and should occasionally be used, glucocorticoids should always be the primary weapon in the treatment of uveitis. The potency of corticosteroid treatment depends on the type of drug used, the route of administration and the daily dosage. The route of administration of choice in iridocyclitis is topical because it allows good drug penetration in the anterior uveal tract while minimizing systemic side-effects. The next choice is bulbar subconjunctival injection which gives a high drug concentration in the iris, ciliary body and aqueous humor. The third most commonly used mode of administration is the systemic route. Because of the possible side-effects, this route should be reserved for severe cases or in instances where other methods of treatment are impractical. The type of glucocorticoid used topically is another very important factor since the degree of anti-inflammatory activity and of intraocular penetration varies markedly from one steroid to the other. For example, hydrocortisone is considered to be a weak steroid compared to dexamethasone or fluorometholone and preparations

that are acetate derivatives penetrate the intact cornea more efficiently than alcohols or phosphates.

If a corneal ulcer or erosion is also present, the use of topical or subconjunctival steroids is strongly contraindicated. If the degree of anterior uveitis necessitates steroid treatment, systemic routes are least damaging. The dosage should be maintained as low as possible by also prescribing nonsteroidal anti-inflammatory agents.

Mydriatics

Anticholinergic mydriatics constitute the other absolutely essential part of treatment in anterior uveitis. Mydriatics minimize the extent and the consequences of posterior synechiae.

The other reason to use anticholinergic drugs is to relieve pain. Part of the pain present in anterior uveitis is caused by a spastic contraction of the ciliary muscle. The cycloplegic effect of parasympatholytic drugs will thus make the eye much more comfortable. The general principle involved in the use of anticholinergic drugs is to be initially as aggressive as necessary to obtain mydriasis and then to keep the pupil dilated. Atropine is usually the drug of choice because of its potency and duration.

Others

The systemic use of antibacterial drugs is generally not indicated in the treatment of iridocyclitis unless there has been a corneal or scleral perforation or if a systemic bacterial infection is suspected. In case of mycotic or protozoal infections, the use of the appropriate systemic drugs is essential (3, 7, 8).

Treatment of Posterior Uveitis

Choroiditis can only be effectively treated by systemic administration of drugs since topical medications and subconjunctival injections fail to achieve therapeutic levels in the choroids and retina. Specific treatment is mandatory whenever a specific cause has been identified but, even then, the oral or parenteral use of glucocorticoids is necessary to minimize the degree of permanent scarring and loss of function. The dosage and duration of treatment depend on the extent of the inflammatory reaction and on the response to treatment (1, 3). Pic. 4.



Pic. 4. Dog, Akita Inu with VKH – like syndrome.

Conclusions

Uveitis can range from a mild, innocuous disease to a severely painful condition causing permanent blindness. It becomes essential to establish a morphological diagnosis very promptly if efficient therapeutic measures are to be taken. While the cause of uveitis often remains undetermined, it is imperative to suppress the inflammation with nonspecific treatment before permanent, severe damage has caused irreversible blindness.

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