Introduction: Equine Uveitis is the most common cause of equine blindness and it has an estimated yearly cost to the equine community of 100 to 250 million dollars. The prevalence of equine uveitis has been estimated to be anywhere from 8 to 25% in the general equine population. Overt costs accrue from medications for treatment and veterinary care. Indirect losses result from Additional less tangible costs such as from poor performance, disruption of training, missed competitions and loss of use are an issue as well. Euthanasia for practical and economic reasons is not uncommon. A clear understanding of the disease and client education is of paramount importance to support the well being of the patient.

Classic ERU and Insidious Uveitis: Uveitis can also be divided into two separate syndromes Classic ERU and Insidious Uveitis, which is the form most commonly found in Appaloosas. Cases of classic ERU are characterized by intense bouts of intraocular inflammation followed by periods of clinical quiescence. Research has shown however, that even in the absence of overt inflammation, ocular degeneration is ongoing. The insidious form of uveitis does not show overt painful episodes of inflammation. Subtle signs of low grade chronic intraocular inflammation, superior lid ptosis, increased lacrimation, and miosis is present, the signs of which are often not easily noticed often go unnoticed until the disease is far progressed. The insidious form is common in the Appaloosa horse. Uveitis is considered recurrent in the horse if more than two episodes have been observed to occur. After a single episode of inflammation the risk of recurrent episodes is decreased after two or more years of a disease free state.

Anatomy of Equine Uveitis: The anterior uvea consists of the iris and the ciliary body. While the posterior uvea is made up of the choroid choroids (was that supposed to be 2 sentences or separated by comma or semicolon??). The choroid is the major blood supply to the retina in the horse and this structure lies between the sclera and the retina. In total, the uveal tract contains the majority of the blood supply to the eye and maintenance of ocular health depends upon maintenance of a barrier between the vascular system and the internal ocular environment via the blood ocular barrier. The blood ocular barrier prevents the migration of large molecules into the intraocular environment and maintains the eye’s immune-privileged status.

Ocular Pathology & Equine Uveitis: All cases of uveitis result from are due to damage to the uveal tract that causes release of inflammatory mediators, prostaglandins, leukotrienes and histamines. Inflammation of the uveal tissues manifests as vascular congestion; the dilation of the scleral and conjunctival blood vessels or results in the “ciliary flush” gives an inflamed eye its red appearance. The release of inflammatory mediators causes increased vascular permeability and breakdown of the blood aqueous barrier. The resulting inflammatory mediators cause the ciliary and iris sphincter muscles to spasm resulting in discomfort. These inflammatory responses cause the common clinical signs of uveitis, including blepharospasm, increased lacrimation, aqueous flare, hypopyon, fibrin accumulation and miosis.

Uveitis also frequently causes results in the inflammation of more than just uveal tissues, associated abnormalities of the eyelids, conjunctiva, cornea, lens, retina, and optic nerve. Blepharospasm, increased serous lacrimation to mucopurulent discharge, chemosis and conjunctival hyperemia are typical. Corneal cloudiness or edema is commonly seen. Edema results from inflammatory damage to the corneal endothelial cells resulting of seepage of aqueous into the corneal stroma. Normally, the metabolic pump mechanism within the endothelial cells draws aqueous out of the corneal stroma; this function is diminished in the inflamed eye. In addition to edema, 360 degrees corneal neovascularization, normally a 360º infiltration of neovascularization of the corneal stromal tissue occurs after several days of inflammation.
Aqueous flare is a hallmark of equine uveitis. The accumulation of non-cellular exudate causes the dysfunction of the ciliary body cells, resulting in a reduction in aqueous humor production giving the hypotonia that is found in uveitic eyes. Hypopyon, can be observed with the settling of inflammatory cells into the ventral aspect of the anterior chamber and intraocular bleeding occur in severe cases.

Might be good so even subdivide more into “acute” and “chronic” in some of these sections:

The iris spincter and ciliary body muscles are affected by the inflammatory mediators in uveitis, causing ciliary body and spincter muscles to spasm resulting in marked miosis. The iris can also take on a dull appearance with mottled pigmentation and hyperemia. Chronic cases of uveitis can exhibit atrophy of the granular irides (corpora nigra) and hyper or hypo-pigmentation of the iris tissue.

Early lens opacities in uveitis consist of inflammatory exudate adhering to the lens capsule. Pigment can migrate from the iris and deposit on the anterior lens capsule or posterior synechia of the iris to the lens causing lens opacification. With changes in aqueous humor composition in uveitis, the metabolism of the lens is compromised, and lens transparency is reduced resulting in cataract development. In chronic cases of uveitis the inflammation can cause the premature degeneration or detachment of lens zonules resulting in lens luxation.

Inflammation of the posterior uvea is accompanied by the infiltration of inflammatory cells into the normally acellular vitreous body. The inflammatory infiltrate can give the vitreous body a distinct yellow color. With chronicity, migration of cells into the vitreous can result in fibrous strands of aggregated inflammatory cells and inflammatory products (vitreal traction bands). Vitreal liquefaction occurs and strands of inflammatory debris may appear can be seen to "wave" in the liquid vitreous with globe movement. Vitreal degeneration and vitreal traction bands that physically pull on the retina cause retinal detachment.

Fundic examination in horse with current of past bouts of uveitis can show peri-papillary depigmentation and chorioretinal scaring (butterfly lesions). Inflammatory lesions can also appear as multiple focal circular areas of depigmentation (target lesions). Evidence of retinal degeneration can also present as changes in reflectivity or color of the tapetal fundus. Inflammatory episodes can cause changes in choroidal blood flow causing retinal cellular hypoxia. The influx of infiltrates and exudates from the choroidal vasculature in-to the sub-retinal space results in retinal detachments. Severe choroidal inflammation can cause disruption in blood flow to the optic nerve resulting in optic nerve damage. Additionally, inflammatory cell accumulation within the aqueous filtration angle and uveoscleral outflow channels can result in the development of glaucoma (trying to keep it simpler).

**Etiology of Equine Uveitis & Patho:** There is not a *genesis* single cause of insidious equine uveitis or classic equine recurrent uveitis. Many infectious agents have been implicated, particularly associated with the onset disease, with a great deal of attention to the role of leptospirosis. Leptospira organisms have been shown to persist in the eyes of horses with equine recurrent uveitis, but the organism’s role in the development of recurrent disease has not been clearly identified. Molecular mimicry between leptospiral DNA and proteins of the equine cornea and lens, supports an autoimmune component cause of the disease. Disruption of the blood ocular barrier allows for the activation of host immune responses with the expected production of antibodies to foreign antigens and the inappropriate production of antibodies to self antigens. The pathogenesis of equine recurrent uveitis has clearly been shown to be immune-mediated with a T-helper cell type response. (seems redundant so I deleted it) It is thought that immune-mediated pathologies can occur because antigens of some pathogens are very similar to self antigens. If a susceptible individual is exposed to such a “molecular mimic”, the immune system erroneously reacts against the self antigen as well, leading to tissue injury. The genetic makeup of an individual horse may also play a role in determining its susceptibility to develop insidious or recurrent uveitis.
**Breed Predisposition /Appaloosa Horses:** Research has shown that Appaloosa horses are 8.3 times more likely to develop uveitis than other breeds. Additionally, Appaloosa horses that are seropositive for Leptospira interrogans serovar pomona have more severe signs of disease and close to a 100% occurrence of blindness. Appaloosa horses tend to suffer from the insidious form of the uveitis without overt episodes of inflammation. The pathogenesis of the disease in the Appaloosa horse may be completely distinct from the classic form that is seen in other breeds. Definitive information as to the age of onset and the duration of the syndrome is hard to define in that the signs of disease are often not noticed by owners until the disease is far progressed. Affected horses more commonly have coats with lighter patterns and focal dark spots. Dark colored Appaloosas with a blanket tend not to develop the disease as commonly. Interestingly, a large portion of Appaloosas that are affected with uveitis also show signs of obstructive airway disease. Uveitis in the Appaloosa horse likely has a genetic basis and specific genetic markers have been identified in affected horses.

**Treatment & Management of Equine Uveitis:** The goals of uveitis therapy include the preservation of vision, minimizing ocular damage and providing comfort. Diagnostic testing for specific infectious causes is warranted. Serologic screening for bacterial and viral agents, particularly testing for seroreactivity to Leptospiral interrogans serovars, is recommended based upon physical examination, complete blood count and serum biochemistry findings. If a specific inciting cause can be found specific treatment is indicated in addition of treatment of the intraocular inflammation. (and any resultant secondary glaucoma??).

**Prevention??? Or Vaccinations**

Vaccination with multivalent vaccines or administering multiple vaccines at once has been clinically associated with a recurrence of ocular inflammation. Generally, spacing vaccinations a week apart and limiting the use of unnecessary vaccines based on geographic location and animal use is recommended. Concurrent treatment with systemic or topical anti-inflammatories has been supported by some authors. A recent report looked at the efficacy of vaccinating horses against pathogenic leptospiral serovars and the data did not support the use of leptospiral vaccinations in horses. Although vaccinated horses had a significant increase in the days to recurrence of inflammation, vaccination failed to slow the progression of the disease, and more vaccinated horses experienced progression of the disease than those in the control group (reference??).

Oral and topical anti-inflammatories are the mainstay of uveitis management. The clinical impression has been that flunixin meglumine has better ocular anti-inflammatory effects than phenylbutazone. Topical steroids combined with antibiotics are very helpful in the treatment of acute cases, however their long term use is associated with an increase in bacterial and fungal keratitis. The use of topical non-steroidal is recommended for long term therapy if necessary. Treatment of equine uveitis can be extremely frustrating and sometimes impossible due to the intractable nature of some horses. The development of the cyclosporine implant provides an alternative to consistent topical management. Candidates for implant placement must have no other systemic illnesses, they must have good vision and no cataracts. Horses, development in the eye to be treated, and must also be well controlled with conventional medical treatments. Initial research estimates that the implant can be effective for up to four years. Summary?? The future of ERU?? Or other?? and that the eye will be supplied with immunosuppressive doses of cyclosporine during that time.