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Equine Herpesvirus-Associated Ocular Disease

Herpesviruses are widely disseminated in nature and renowned in equine medicine for inducing respiratory, reproductive, and neurologic diseases.

The most important biological characteristic of these viruses is their ability to remain latent. Reactivation of latent virus can occur and result in virus replication, shedding, and disease. While the precise mechanisms of reactivation are unclear, it is believed to be triggered by stressors such as exercise, pregnancy, changes in management, or concurrent disease. The equine herpesviruses (EHV) that have been implicated in ocular disease, to date, are EHV-2 and EHV-1.

Equine herpesvirus-2 has been proposed as the putative and primary cause of viral keratitis in the horse. Nevertheless, the virus has been inconsistently found in corneal and conjunctival scrapes of affected animals and can be frequently found in asymptomatic horses. In addition, co-infection with EHV-5 is common not only in ocular disease, but also in respiratory cases, making it difficult to attribute causation of disease to a particular virus.

Evidence for EHV-2 induced ocular disease is based on several reports of successful virus isolation from outbreaks and single cases of keratoconjunctivitis (inflammation of the cornea and conjunctiva), conjunctivitis (inflammation of the conjunctiva), or corneal edema (swelling of the cornea) in foals; however, despite such reports its role as a pathogen is still debatable. In contrast to other viral etiologies, herpetic keratitis is not usually accompanied by systemic disease. Clinically, horses have sporadic, multifocal, subepithelial, punctate opacities (areas of cloudiness) that stain variably with fluorescein but commonly with Rose Bengal. Superficial vascularization is associated with the opacities, and horses exhibit persistent to moderate ocular pain that can lead to reflex uveitis (inflammation of the uvea). Continuation of the epithelial lesions into the corneal stroma is possible as a consequence of viral antigens extending from epithelial lesions or from viral replication in stromal cells.

Evidence of virus in corneal and/or conjunctival scrapes can be challenging as viral inclusion bodies, within injured epithelium, are rarely seen at the time of sampling. PCR techniques are more reliable in detecting viral DNA; however, the presence of herpetic DNA is not diagnostic of itself.

Corneal lesions in the horse seem to resolve quickly after antiviral treatment or heal spontaneously, which is in contrast to human and feline induced herpesviral ocular disease. Treatment with topical antivirals is often recommended if there is a strong suspicion of herpesvirus involvement and subsequent PCR confirmation. Topical ganciclovir gel in combination with topical corticosteroids has been recommended as the treatment of choice.

Chorioretinal lesions (those in the eye's choroid and retina) have been described following experimental EHV-1 infection in several studies. Focal and multifocal lesions, either unilateral or bilateral and located in the non-tapetal fundus as donut-shaped depigmented regions with pigmented centers were observed several weeks after infection. This type of lesion is most likely caused by ischemic injury of the chorioretina with death of the overlying retinal pigment epithelium. These findings do not seem to impact vision, but are a main concern during pre-purchase examination, particularly if previous history is unknown.

Although there is sufficient evidence to support the role of EHV-1 in chorioretinitis, the implication of any member of the equine herpesvirus family in corneal disease remains controversial.