CANINE HERPESVIRUS

By Leslie Manis, Health/Genetics Chairman, ASTC

On October 23-25, I attended the Biennial AKC Canine Health Foundation 2009 National Parent Club Canine Health Conference. As always, it was a very informative and stimulating event, with speakers on a variety of subjects as well as round table discussions on topics such as parent club health surveys. This article is from a series I wrote summarizing information on various health topics presented at the conference.

Presentation Summary:

Canine Herpesvirus-1: A New Pathogenic Role for an Old Virus by Eric C. Ledbetter, DVM, DACVO, Cornell University

Canine herpesvirus-1 (CHV-1) was first identified in the mid-1960s as a cause of severe morbidity & mortality in fetal and neonatal dogs. In the following decades, CHV-1 infections in mature dogs were sporadically and infrequently associated with several relatively mild conditions, including genital mucositis and respiratory tract disease. More recently,the significance of CHV-1 as an ocular pathogen in mature dogs has been recognized and investigated.

In the past few years, several ocular diseases have been linked to primary and recurrent CHV1 infection in mature dogs, including conjunctivitis, ulcerative keratitis (scratched cornea) and non-ulcerative keratitis. In addition, CHV-1 has been reported as the most common cause of viral conjunctivitis in client-owned dogs and as a cause of large outbreaks of ocular disease in group-housed dogs.

The true prevalence of CHV- 1 ocular diseases remains unknown, but recent discoveries suggest a common and substantial role for this virus in the development of a variety of ocular diseases in dogs of all ages.

Canine herpesvirus-1 is among the most prevalent infectious diseases in dogs worldwide. In fact, it is possibly the most prevalent. It affects domestic dogs and their wild cousins & is related to herpes simplex virus-1 & -2 in humans, feline herpesvirus-1 and bovine herpesvirus-1.

Severity of disease depends upon the age of the host; it is most severe in the very young and immunosuppressed dogs (those with lymphoma for example) and localized in the mature dogs, affecting the eye, mouth, respiratory system or genitals.

These herpes viruses are associated with recurrent eye disease in their respective host species. During the initial ocular infection, the virus travels through the sensory nerves and becomes latent in the ocular sensory ganglia. Clinically, there is no obvious difference between viral conjunctivitis and other causes. Latent herpes infection may reactivate spontaneously or as a result of use of corticosteroid, cyclosporine (as in dry eye

medication), cyclophosphamide or epinephrine administration, also eye surgery or trauma, stress or fever. In a bitch that has never been exposed to canine herpes, then acquires it during pregnancy, the virus will travel through the placenta to the fetus. The puppies can be reabsorbed, aborted or born prematurely.

Puppies can get it in utero, in the birth canal or from littermates or the dam. This was first identified in the '60s and called fading puppy syndrome. It often affects puppies less than two weeks old and is fatal.

In 2006 were. the first two confirmed cases of CHV-1 ocular disease. They were able to isolate the virus from the eyes of the affected dogs & found it was the same canine herpes virus as the one discovered in the 1960s.

Recurrent infections of adult dogs and the stimuli that contribute to viral reactivation have not been studied extensively prior to Dr. Ledbetter's project. He had three groups of dogs: one group was latently infected with canine herpes then given prednisolone for seven days, group 2 latently infected but given a placebo for seven days and group 3 not latently infected with herpes but given prednisolone.

He then studied the presence (or absence) of ocular disease, viral shedding and antibody titers over time in each group. Eighty-three percent of the dogs in group 1 developed ocular disease an average of 9 days after administration of the steroid. Fifty percent of group 1 had ocular viral shedding.

Fourfold elevations in titers for CHV-1 were detected in 100% of group 1 dogs by day 14. Dogs in control groups did not develop clinical ocular disease, CHV-1 titer elevations or viral shedding. What was disturbing to me was that these high titers in group 1 dropped in 6-8 weeks, making it very hard to determine if the dog was ever exposed.

As a result of this study, recurrent CHV-1 ocular disease should be considered a potential adverse effect of immunosuppressive prednisolone treatment in latently infected adult dogs.

Research to determine the prevalence and clinical significance of naturally acquired recurrent CHV-1 disease in adult dogs is warranted. Perhaps someday, better detection of a dog's exposure to the virus and even the development of a vaccine could become a reality.

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