

Canine Distemper in Wildlife: How Private Practitioners Can Help

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THE CANINE DISTEMPER VIRUS AND ITS RELATIVES:

Canine distemper virus (CDV) is a morbillivirus (family *Paramyxovirinae*) and is closely related to viruses that cause diseases like measles, peste des petits, and the recently eradicated Rinderpest. Dolphin morbillivirus and phocine distemper virus also are closely related. All are non-segmented negative sense single-stranded RNA viruses. But let's be honest, why would anybody but a virologist care if CDV is a RNA or a DNA virus? Probably the most important reason for the practicing veterinarian to remember it is an RNA virus is because an RNA virus like CDV does not have a proofreading mechanism for replication (DNA viruses have DNA polymerase). This permits or facilitates rapid viral mutation. The story of CDV is one of viral mutation and an ever-expanding host range.

HOST RANGE:

While domestic and wild canids play a major role in the transmission of CDV worldwide, numerous other species have been infected with and are susceptible to CDV.^{1,8} As evidenced by clinical disease or antibody titers from free-ranging or captive wild animals, susceptible terrestrial carnivore families in addition to canines, include Felidae (African lion, tiger, leopard, jaguar, etc.), Hyaenidae (e.g. spotted hyena), Mustelidae (e.g. domestic ferret, black-footed ferret, North American river otter, etc.), Procyonidae (e.g. raccoon and kinkajou), Ursidae (e.g. black bear, brown bear, etc.), and Viverridae (e.g. binturong and palm civet).¹ Also, two seal species, Baikal³ and Caspian⁴ seals, have experienced CDV epizootics. Epizootics also have been documented in collared peccaries and serology suggests that the virus could be enzootic in peccary populations in southern Arizona.

VIRAL TRANSMISSION AND EPIDEMIOLOGY:

Canine distemper virus is epitheliotropic, replicating rapidly in epithelial cells. Consequently, major modes of transmission include aerosolization of virus through respiratory exudate, and probably to a lesser extent through oral secretions, urine and feces. The virus is relatively short-lived in the environment and transmission is usually through close contact. Animals will begin to shed virus 7 days post infection and may continue to shed for 60-90 days. There is no carrier state and once infected, animals will either survive, cease viral shedding and become immune, or die.⁸ Therefore, viral maintenance requires an ever-continuing supply of susceptible hosts occurring at high density. Infection is said to move through populations much as a fire will burn a forest. Just as an area of forest will burn and then cannot burn again until new "fuel" is available, an epizootic of CDV will pass through a dense population of susceptible animals (that will individually die or become immune) and the virus will be unable to return until new animals susceptible to CDV enter the population, either through birth or immigration. This can be seen when looking at domestic dog populations that are not vaccinated. In endemic areas where the virus continuously cycles, clinical disease in dogs is mostly seen in pups that have lost maternal antibodies because older animals have already been exposed to CDV and are resistant (or they have become infected and died). In epizootic areas where outbreaks occur randomly, they often are widespread and affect dogs of all ages (because all animals in the population are susceptible).

CLINICAL SIGNS, & PATHOGENESIS:

In addition to host susceptibility (for example, gray foxes are highly susceptible to CDV, whereas red foxes can be infected, but seem more resistant), clinical signs of CDV infection are modulated by viral virulence, environmental conditions, and host immunity. Major organ systems affected include the respiratory, gastrointestinal, integumentary, and central nervous systems. While pathogenesis of CDV infection in most wild animals has not been well studied, it has been studied in domestic canids, mink, and ferrets. Virus usually enters via the epithelium of the upper respiratory tract, multiplies in macrophages and spreads to tonsils and regional lymph nodes, where viral replication can occur within 2 to 4 days post-infection. Within a week, CDV proliferates in lymphoid organs such as the spleen, mesenteric lymph nodes, Kupffer's cells in the liver, and the lamina propria of the stomach and small intestine. Fever and leukopenia are associated with viral spread due to loss of T and B cells. Eventually virus spreads to epithelial cells throughout the body. Dogs with adequate humoral and cellular immunity might show clinical signs, but will clear the virus from most tissues within 3 weeks. Possible exceptions might include the CNS, lung and skin where virus can be shed for several months. If there is inadequate immune response, severe clinical disease is seen at 2-3 weeks with death by 3-4 weeks. Dogs that recover can shed virus for 2-3 months.^{1,8}

In some wildlife species, it has been suggested that concurrent bacterial infections can exacerbate clinical disease caused by CDV and it is possible that high levels of organic pollutants also could increase disease susceptibility.

DIAGNOSIS

History, clinical signs and typical gross lesions can suggest CDV infection. Common microscopic lesions include depletion of lymphocytes in paracortical zones and germinal centers of lymph nodes and spleen and thymic atrophy, interstitial pneumonia, nonsuppurative encephalitis and meningoencephalitis, neuronal necrosis and focal malacia and demyelination in cerebellar white matter. Intracytoplasmic and intranuclear eosinophilic inclusion bodies in epithelia, neurons and astroglia are common. They can also be seen in gastric mucosa, pancreatic and biliary duct epithelium, epithelium of the respiratory tract and in transitional epithelium of the bladder. Because of the immunosuppressive effects of CDV on the host, wildlife can present with concurrent infections that can complicate diagnosis. These include secondary bacterial or protozoal infections like toxoplasmosis, encephalitozoonosis and coccidiosis. Concurrent infections with parvovirus or rabies can occur with CDV infection and should not be overlooked.⁸

Definitive diagnosis of CDV infection can be made with Fluorescent Antibody (FA) staining, immunohistochemistry, viral isolation, or rt-PCR. Note that serology should not be used for definitive diagnosis. In some cases, animals that die of CDV do not mount an immune response and in dogs, serum neutralization antibody titers vary inversely with disease severity.

SPILLOVER FROM DOMESTIC ANIMALS TO WILDLIFE

There are multiple documented cases where CDV has been transmitted from domestic dogs to wildlife and additional instances where CDV in wildlife is believed to have come from domestic dogs. Both of the 1987 and 2000 CDV epizootics in Baikal and Caspian seals, respectively, likely originated from CDV epizootics in domestic dogs.^{3,4} Viral homology of the CDV H gene in a CDV-infected wild wolf and domestic dog suggest that domestic dogs were responsible for transmitting CDV to wild wolves in Portugal in 2007-2008.⁶ Between 2001 and 2003 an epizootic of CDV in black-backed jackals and other wild carnivores in Namibia was attributed to domestic dogs based on viral sequence data from the P and H genes.² A CDV epizootic in domestic dogs in Kenya between 1990 and 1992 is believed to have been responsible for the disappearance of known packs of African wild dogs in the region.⁷ Also, it has been hypothesized that domestic dogs could have transmitted CDV to wild giant pandas in Wolong Reserve, China.⁵

VACCINE-INDUCED DISEASE IN WILDLIFE

Vaccine-induced canine distemper has been demonstrated in numerous wildlife species including the African wild dog, black-footed ferret, kinkajou, lesser panda, maned wolf, and gray fox. Suspected vaccine-induced canine distemper has occurred in raccoons, fennec fox, and the South American bush dog. All have been associated with administration of various modified live vaccines.

THE PRACTICING VETERINARIAN'S ROLE IN CONTROLLING CANINE DISTEMPER VIRUS:

The numerous cases of documented and suspected transmission of CDV from domestic dogs to wildlife is a stark reminder that prevention of disease in domestic animals is an important tool in wildlife health. Rinderpest, a morbillivirus closely related to CDV, circulated between wild and domestic animals in Africa and Asia causing massive mortality in both. When a vaccine was developed, there was fear that Rinderpest would never be eradicated because of its sylvatic cycle. Massive vaccination of domestic livestock, however, removed enough of the susceptible population to enable disease eradication without ever vaccinating wildlife. While this may or may not be possible for CDV, practicing veterinarians can help prevent CDV epizootics in wildlife by vaccinating domestic dogs for CDV. Massive and sustained CDV vaccination campaigns in domestic dogs have the potential to rapidly protect highly endangered species such as tigers, African wild dogs, and Ethiopian wolves. Widespread companion animal vaccination also will benefit non-endangered susceptible wildlife as well.

Veterinarians working in zoological or wildlife rehabilitation settings should be reminded of the numerous wildlife species in which CDV vaccination has caused CDV infection, and in some cases, started CDV epizootics in free-ranging wildlife. If the decision is made to vaccinate a wild animal, veterinarians should consider using a recombinant canary pox CDV vaccine where the CDV F and HA proteins are inserted into the canary pox viral genome and vaccination against CDV enables the canary pox virus to enter the host's cell where the DNA is transcribed creating the F and HA proteins. These are taken by macrophages and presented to lymphocytes resulting in CDV immunity. With the use of such a vaccine, there is no opportunity for vaccine-induced CDV.

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