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Canine Distemper, A Review

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Abstract

Canine distemper (CD) is acute, highly infectious viral disease and the most important worldwide domestic dogs disease (Canis familiars), and its fatality rate is second only to that of rabies affect carnivores and domestic dogs of any age. Canine distemper virus, a relatively large (150–250 nm) single-stranded RNA virus with a lipoprotein envelope, is a Morbillivirus in the family Paramyxoviridae. Three other well-known diseases are caused by members of the Morbillivirus genus: Measles in primates, Rinder pest in artiodactyls, and pestedespetits ruminants in small ruminants. Canine distemper virus can infect a wide range of carnivore's including members of the Canidae, Procyonidae and Mustelidae. Canine distemper is common in young unvaccinated dogs, usually in their first year of life. The transmission is through aerosolization of respiratory exudates containing virus, although other body excretions and secretions (e.g., urine) can result in infection in susceptible hosts if aerosolized and transmitted by direct contact. There is no curative treatment for canine distemper infection; treatment is mostly supportive and for secondary complication and preventedby vaccination. Ethiopia suffered from the diseases in different parts of the country especially the Bale mountain national park wolves which attract tour are at risk. Awareness about these diseases should be created and vaccination by using attenuated vaccine should be advocated.

Keywords: Canine, Dogs, Distemper, Infectious

Introduction

Canine distemper isacute, highly infectious viral disease and the most important worldwide disease of domestic dogs (Canis familiars), and its fatality rate is second only to that of rabies affect carnivores and domestic dogs of any age (Swango *et al*., 1995). CD is caused by canine distemper virus (CDV), first isolated in 1905(Carre, 1905). It is a highly contagious viral pathogen causing lethal disease in both domestic and wild, land and sea-living animals. It is classified in the Morbillivirus genus of the family Paramyxoviridae (Griffin, 2001; Murphy *et al.*, 1999).The Morbillivirus genus includes other important highly infectious pathogens like Measles Virus (MV), Rinderpest Viruses (RPV), and almost all members' present equivalent tropism and tissue distribution in their respective hosts. Morbillivirusis transmitted by aerosols, direct contact, and produce clinical similarities, such as fever, serous nasal discharge, and cough, as well as respiratory and gastrointestinal signs often complicated by secondary bacterial infections. Furthermore, the most notorious property of Morbillivirus infection is the establishing of severe

transitory immunosuppression (Messling *et al*., 2003; Griffin, 2007).

Canine distemper virus (CDV) can infect a wide range of carnivore including members of the Canidae, Procyonidae,and Mustelidae. CD is common in young unvaccinated dogs, usually in their first year of life, but many cases are also seen in adults (Tipold, *et al.*, 1992).Canine distemper causes some of the highest mortality rates for canine infectious disease (Shell, 1990; Appel and Summers, 1995).Puppies with distemper develop pneumonia, conjunctivitis, rhinitis, and tracheitis. The lungs are typically edematous, and microscopically there is broncho-interstitial pneumonia with necrosis of the epithelium lining the small airways and thickening of the alveolar walls (McLachlan and Dubovi, 2011).There is no specific treatment for canine distemper and prevented by vaccination. A number of vaccines against canine distemper are available for dogs and other domestic and nondomestic animals (Deem *et al*., 2000). CD in dog and Ethiopian wolves can play a pivot role in dynamic of endangered species and population; the virus can affect the size and viability of population both directly through effects on hosts survive and reproduction but also indirectly by altering their behavior, movement patterns, social system and community structure (Sillero *et al*., 1996).Therefore, the objective of this seminar paper is:

- To review the etiology, epidemiology and to summarize methods of prevention and control of canine distemper virus.

Canine distemper

Etiology

CDV, a relatively large (150–250 nm) single-stranded RNA virus with a lipoprotein envelope, is a Morbillivirus in the family Paramyxoviridae (Greene and apple, 1990; Swango *et al.*, 1995) three other well-known diseases are caused by members of the Morbillivirus genus: Measles in primates, Rinderpest in artiodactyls, and Peste des petit ruminants in small ruminants. Three recently discovered viruses, phocine distemper virus in seals and cetacean morbillivirus in porpoises and dolphins, also belong to this genus (Osterhaus *et al.*, 1995; Visser *et al.*, 1993).

Epidemiology

Geographic distribution and Host susceptibility

CDV is a worldwide distribution that occurs in all members of the Canidae, Mustelidae, and Procyonidae families (APPLE, 1987). CD is common in young unvaccinated dogs, usually in their first year of life, but many cases are also seen in adults (Tipold *et al.*, 1992).In addition host susceptibility (for example, gray foxes are highly susceptible to CDV, whereas red foxes can be infected, but seems more resistant). (Deem *et al*, 2000, Williams, 2001). The disease has been reported in Asian elephants, Elephas Maximus (Creevy, 2013).

Mode of transmission and source of infection

The most common source of infection is direct contact between the susceptible dog and infected dogs or wildlife. The major mode of CDV transmission is through aerosolization of respiratory exudate containing the virus, although other body excretions and secretions (e.g., urine) can result in infection in susceptible hosts if aerosolized. It's IP ranges from about 1 wks to 1 month. Canine distemper is highly contagious, and viral shedding may follow infection for 60–90 days (Greene *et al*., 1990).Transplacental infection has been documented in domestic dogs (Krakowka *et al.*, 1997).The epidemiologic role of vertical transmission in CD and whether or not such transmission can occur in nondomestic species are unknown. Although usually short-lived in the environment, the virus can survive at lower temperatures (e.g, 48 hr at 25^{o}c and 14 days at 5^{o}c). Also transmitted either by direct contact or by fomites (Pearson and Gorham, 1987).

Pathogenesis

CD pathogenesis in domestic dogs has been well characterized and may be similar in non-domestic species. The 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 leucopenia 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 the 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. (Deem *et al.*, 2000 and Williams, 2001)

The pathogenesis within 9–14 days depends on the humoral and cell-mediated host immune response. Dogs with the neurologic disease may develop hyperkeratosis (thickening) of the footpads and nose as a result of epithelial damage caused by the virus. This manifestation in dogs gave rise to the term "**hardpad disease**" as an alternative common name for canine distemper (see in figure 1). In dogs, less than half of the adult animals that are infected with CDV die from it; among puppies, however, the death rate can be as high as 80% (Appel,1987; Greeneand Appel, 1990).

Clinical signs

Clinical signs of CDV infection are modulated by viral virulence, environmental conditions, and hostage and immunity. Major organ systems affected include the respiratory, gastrointestinal, integumentary, and central nervous systems. Diphasic fever and general malaise are often associated with viremia. Infections, probably secondary to leukopenia, are common and may complicate the clinical course.(Deem, *et al*, 2000; Williams, 2001). CD is related to the respiratory and gastrointestinal systems and includes conjunctivitis, pneumonia, diarrhea (often hemorrhagic), anorexia, and severe dehydration. A neurologic manifestation of CD may occur 1–3 wk after recovery from acute generalized infection (Appel, 1987;Vandervelde and Cachin, 1992).

Neurologic distemper can occur in dogs of any age that had no or mild systemic signs and may manifest as chronic progressive neurologic dysfunction in older dogs (usually over 6 years of age). Neurologic complications depend on viral distribution in the CNS and may include hyperesthesia, cervical rigidity, seizures, cerebellar and vestibular signs, and paraparesis ortetraparesis with sensory ataxia. Myoclonus, the involuntary twitching of muscles in a forceful simultaneous contraction (often leading to ‘‘chewing gum’’ fits), is also highly suggestive of CD and Additional signs of CDV in the domestic dog include digital hyperkeratosis (hard pads) and optic neuritis, chorioretinitis, and uveitis (Gelatt *et al.*,1985).Puppies with distemper develop pneumonia, conjunctivitis, rhinitis, and tracheitis. The lungs are typically edematous, and microscopically there is broncho-interstitial pneumonia with necrosis of the epithelium lining the small airways and thickening of the alveolar walls (McLachlan and Dubovi, 2011).

Diagnosis

Physical Diagnosis

In domestic dogs, acute generalized CD infection is often diagnosed by history, clinical signs in animals not previously vaccinated. In nondomestic species, the CD may be suspected on the basis of clinical signs but must be differentiated from such other diseases with respiratory, neurologic, and/or gastrointestinal manifestations as rabies, feline pan leukopenia, toxoplasmosis, canine parvovirus, lead poisoning, and bacterial entreaties. Digital nasal, and eyelid hyperkeratosis, common in infected ferrets and mink(Davidson, 1986; Pearson and Gorham, 1987). Are highly suggestive of infection in raccoons, foxes, and ferrets, jaundice associated with CDV infection is occasional and unique (Budd, 1981).

Laboratory finding

Laboratory tests are necessary for a definitive diagnosis and to exclude other diseases with similar clinical manifestations While virus isolation is possible, it is laborious as it requires co-cultivation of lymphocytes from the suspect animal with cell lines expressing the receptor molecule (MacLachlan and Dubovi, 2011). Absolute lymphopenia, regenerative anemia, decreased albumin, and increased α-and γ-globulin concentrations may be present(Greene and Appel 1990; Swango, 1995).

Low numbers of CDV inclusions may be detected in the cytoplasm (and occasionally nuclei) of stained peripheral blood cells, especially lymphocytes. Inclusion bodies may also be detected in smears prepared from conjunctival scrapings. However, inclusion bodies are unlikely to be present in either theblood or conjunctival scrapings outside of the acutephase of infection. Interstitial or alveolar lung patterns on thoracic radiographs also support a diagnosis. Central spinal fluid (CSF) may show increased protein (>25 mg/dl) and cell count (>10

cells/ml with a predominance of lymphocytes) and increased pressure associated with inflammation. Increased anti-CDV antibody in the CSF is definitive evidence of neurologic CDV infection. Unless the blood-brain barrier has been disrupted because CDV specific IgG is not present in the CSF of vaccinated dogs (Johnson *et al*., 1994).

Enzyme-linked immune sorbent assays (ELISA) have been developed to detect serum IgG and IgM antibodies to CDV (Noon *et al.,* 1980; Potgieter and Ajidagba 1989; Von Messling *et al.*,1999). The detection of IgG is more ambiguous and can indicate either vaccination or infection. Immunohistocytochemistry is also useful in diagnosing CD, Immunofluorescence test (IFT) is usually performed on cytology smears prepared fro mthe conjunctival, tonsillar, genital,respiratory epithelium(Axthelm and Krakowka, 1986).It is difficult to isolate CDV by routine cell culture. Virus isolation is most successful by direct cultivation of target tissues of lymphocytes and macrophages from the infected host. In cultures with no cytopathic effects after 48–72 hr, fluorescent antibody tests can detect CDV (Appel *et al.,* 1992; Appel,1987)and Polymerase chain reaction should be considered for the detection and differentiation of CD (Haas *et al,* 1991).

Postmortem finding

Lesions of CDV infection are similar in nondomestic carnivores and in domestic dogs (Deem *et al*., 2000). The most significant gross lesions are pneu monia depletion of lymphopoietic organs, and hyperkeratosis of the nose, foot pads, and eyelids. In uncomplicated CDV infection, the only consistent pathologic finding is thymic atrophy(Appel *et al,*. 1994).Common histologic findings are hyperkeratosis of the nose, foot pads, and eyelids; eosinophilic inclusion bodies in many organs (most commonly cytoplasmic but occasionally intranuclear in the CNS, urinary bladder, and bronchial epithelium) ; lymphoid depletion; diffuse interstitial pneumonia, a nd perivascular lymphoplasmacytic infiltration in areas of demyelination and neuronal degeneration of the CNS. Syncytial giant cells in the lungs and CNS white matter, anterior uvea, and lymph nodes may also be present. In contrast to histopathology lesions identified in the domestic dog, lungs of large felids may show diffuse alveolar type 2 cell hyperplasia with intracytoplasmic and intranuclear viral inclusion bodies. These cells were strongly positive for CDV antigens by immunohistocytochemical staining.

This cellular response appears to be unique to large felids (Appel *et al.,* 1994). Additionally; feline brain histopathology may lack the typical canids' pattern of demyelination with astrocytosis and vascular cuffing. Most cats have had mild, patchy CNS lesions compared with those of canids. The lung, liver, lymph nodes, brain, and spleen of any dead animal with suspected CDV infection should be collected for viral isolation and/or PCR. Immunohistocytochemistry on formalin-fixed tissues provides definitive evidence of CDV infection (Axthelm and Krakowka, 1986).

Treatment

There is no specific treatment for canine distemper .but they are symptomatic and supportive, aimed at limiting secondary bacterial invasion, supporting fluid balance, and controlling neurologic manifestations. Broad-spectrum antibiotics, balanced electrolyte solutions, parenteral nutrition, antipyretics, analgesics, and anticonvulsants are used, and good nursing care is essential (Creevy, 2013).

Prevention and control

Canine distemper is best prevented by vaccination. A number of vaccines against canine distemper are available for dogs and other domestic and nondomestic animals (Deem *et al.,* 2000). Currently, domestic dogs are vaccinated with commercially available vaccines containing the modified live virus (MLV). These viruses are attenuated by serial passage in tissue culture. Successful immunization of pups with MLV depends on the lack of maternal antibody interference. To attempt to overcome this issue, pups are vaccinated with MLV vaccine at 6 weeks old and at 3- to 4-week intervals until 16 weeks old (Creevy, 2013).

Many varieties of MLV vaccine are available and should be used according to the manufacturers' directions. MLV vaccines can produce disease in some immunosuppressed dogs. In addition, nondomestic species are often more susceptible to CDV than dogs. Thus, standard attenuated virus vaccines should not be used in nondomestic species. A recombinant canarypoxvirus-vectored vaccine is now available that is safe, does not contain live virus, and cannot cause distemper (Pardo *et al*., 1997).The virus is fragile and extremely susceptible to ultraviolet light, heat, desiccation, and common disinfectants such as formaldehyde, phenolic compounds, and quaternary

ammonium compounds. Also, it does not survive in the environment for more than a few hours at room temperature (~25°C); it can though survive for at least two weeks in shady environments at near-refrigeration temperatures (~5°C). Infected animals should be quarantined from other animals for several months due to the possibility of prolonged viral shedding during this time(Shen and Gorham, 1980).

Canine distemper infection in Ethiopia

The Ethiopian wolf (*Canis simensis*) is recognized as the rarest canid species in the world and as the most threatened carnivore in Africa. Fewer than 500 adult and sub-adult wolves remain in half a dozen suitable Afro alpine habitat ranges (International Union for the Conservation of Nature; 2011). The largest population is in the Bale Mountains National Park (BMNP) in southeastern Ethiopia, where wolf populations reach densities of up to 1.4 adults and sub-adults/km2 (Marino *et al*., 2013).

On average, family packs contain 6 adult and sub-adults (range 2–20) and protect a home range of ≈6 km2 (Sillero *et al*.,2004). Such high wolf densities, large packs, and intense social behaviors increase the risks for disease transmission (Sterne and Smith, 2006). As a result of rabies outbreaks during 1991–1992 (Sillero *et al.,* 1996; 2003 Randall *et al*., 2004; 2008–2009 (Johnson *et al*., 2010).Wolf subpopulations in (BNMP) were dramatically reduced by 45%-75%. Serologic evidence for CDV within wolf populations has been reported(Laurenson *et al*., 1998) of 30 samples tested during 1989–1992, a total of 9 (30%) were seropositive for CDV.CD in dogs and Ethiopian wolves can play a pivot role in dynamic of endangered species and population; the virus can affect the size and viability of population both directly through effects on hosts survive and reproduction but also indirectly by altering their behavior, movement patterns, social system and community structure (sillero *et al*., 1996).

Zoonotic potential and future directions

While there is currently no definitive evidence of naturally acquired human CDV infection, there are several reasons to monitor the situation. CDV has a wide host range and appears to have recently adapted to nonhuman primates as evidenced by several large outbreaks among multiple species (Qiu*et al.,* 2011; Sakai *et al.,* 2013; Sun *et al.,* 2010). Furthermore, it has been demonstrated that human cell surface molecules can serve as viral entry receptors for CDV (Bieringer *et al*., 2013; Otsuki *et al.,* 2013). Lastly, due to the close relationship and immunological cross-reactivity with measles virus (MV), some have suggested that when measles vaccination is stopped (due to the eventual eradication of MV), CDV might cross the species barrier to humans and emerge as a new human pathogen. Thus, it is prudent to be prepared with appropriate vaccines and therapeutics for use in humans in the event that CDV becomes a serious human pathogen (Bieringer *et al*., 2013).We must be cautious when working with animal potentially infected with morbillivirus. Potential new vaccines, including DNA (plasmid products against these agents may hold great promise as safe and effective vaccines for exotic carnivores (Sixt *et al*, 1998).

Conclusion and Recommendation

Canine distemper (CD) is the most important worldwide infectious disease of domestic dogs (*Canis familiaris*), and its fatality rate is second only to that of rabies and affect carnivores and domestic dogs of any age, incidence of clinical diseases is almost entirely in puppies between weaning and 6 months of age that has lost its maternal protection but acquired active immunity. The virus causes a high incidence of mortality and morbidity in some breeds of dogs, young aged and immune compromised animal. The absence of curative therapy and its genetic variation make the canine distemper infection the highest clinical diseases of the dog and other canids. In Ethiopia, In addition to being a risk for dogsit is a potential threat for the indigenous Ethiopian wolves in areas where domestic dogs are living in close proximity to wolves' habitat. Based on the above conclusion the following recommendations have been forwarded;

- Canine distemper infection has no curative treatment, therefore, to prevent dogs and other canids from this specific viral infection, vaccination using attenuated live virus vaccine should be advocated in endemic areas.
- Transmission is through aerosolization of respiratory exudate containing the virus, although other body excretions and secretions. Therefore, environmental hygiene where dogs kept should be strictly controlled.
- Emphasis should be given for susceptible groups of dogs during control and prevention of CDV infection in endemic areas.

➢ In Ethiopia, CDV infection is a potential threat for the indigenous Ethiopian wolves in areas where domestic dogs are living in close proximity to wolves ' habitat, therefor, control of disease in da og should be practiced to avoid transmission of disease from dogs to wolves.

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