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Review

Canine Distemper - Review

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Abstract

Canine distemper (CD), is an acute, highly infectious viral disease that is caused by Canine Distemper Virus (CDV) characterized by abdominal pain, diarrhea, and vomiting. The disease results in the rapid onset of severe leucopenia and loss of lymphocyte proliferation ability resulting in immunosuppression in domestic dogs and many other carnivores. It is distributed worldwide and results in high morbidity and mortality. The most common source of CDV infection is direct contact between susceptible dogs and infected dogs or wildlife. CDV is commonly transmitted by aerosol or droplet exposure from respiratory exudates; however, urine and other secretions also contain an infectious virus. The best way to prevent CD is through Vaccination. This review primarily focuses on the history, host susceptibility, structure, transmission clinical signs, diagnosis, and prevention of CDV.

Keywords: Canine Distemper, Gasteroenteritis, Leucopenia, Vaccination.

Introduction

Of all the domesticated animals, dogs have been the best companion animals of all ages. However, they are prone to several bacterial and viral diseases, most of which cause gastroenteritis. Gastroenteritis refers to inflammation of the gastrointestinal tract, meaning the stomach and the intestines. It can be challenging for the veterinarian to determine the actual causative agent responsible for diarrhea due to the diverse array of pathogenic agents that could be responsible including viruses, bacteria, or protozoans. Reportedly, the viruses are the common cause with one or more detected in 40–60% of cases of gastroenteritis in dogs (Alves *et al*., 2018). Gastrointestinal disorders are frequently reported in companion animal clinics as leading to severe dehydration and death. The condition often causes abdominal pain, diarrhea, and vomiting. Viruses associated with enteric illnesses in dogs are an important cause of mortality in non-protected populations ((Decaro and Buonavoglia, 2012). CDV is least suspected for gastroenteritis as CDV infection mainly results in respiratory signs. CDV belongs to the genus *Morbillivirus* within the family *Paramyxoviridae* (Griffin, 2001 and Murphy *et al.*, 1999) and order *Mononegavirales.* CDV is a single-stranded, negative-sense, non-segmented, enveloped RNA virus with a diameter of about 150-300 nm. The genome of CDV is about 15.7 kb. The genome is of negative-sense, single-stranded RNA encoding for six structural proteins; nucleocapsid (N), matrix (M), fusion (F), haemagglutinin (H), polymerase (L), and phosphoprotein (P) (Messling *et al.*, 2001). The disease was first reported in Europe in 1790 (Appel and Gillespie, 1972) and the virus was isolated by Carre. 1905.

Morbillivirus is transmitted by aerosols, and direct contact, and produces clinical signs, 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 establishment of severe transitory immunosuppression (Messling *et al*., 2003 and Nathanson & Griffin. 2007).

The disease has been controlled by the use of attenuated live virus vaccines. However, several CDV-vaccinated dogs also come down with CDV worldwide (Moller *et al.*, 1992). These findings suggested that there are genomic/virulence differences between the wild-type CDV and vaccine strains (Harder and Osterhaus, 1997). Analysis of viral genes provides insights into the understanding of the variations of CDV.

History

CDV is the causative agent of CD which is an acute, highly infectious viral disease (Norris *et al.,* 2006) and the most important disease of domestic dogs (Cleaveland *et al.*, 2006) The disease was imported from South America around 1760 (Blancou, 2004). Edward Jenner first described the course and clinical signs of the disease in 1809 (Shell, 1990). The CD was first described in Spain in 1791 and was first isolated by Carre. H, 1905 (Appel and Gillespie, 1972), hence the disease is still called Carre's disease and over time, it has been reported in a broad range of terrestrial and aquatic carnivores as well as in non-carnivore species.

The seroprevalence of CDV infection has been reported as 70% in South India (Latha *et al*., 2007). She screened 160 conjunctival samples collected from dogs with clinical symptoms suggestive of CD using dot ELISA and 112 (70%) were positive. However, no virus isolation has been done from India until 2010.

Ramanathan *et al.* (2007) found 49 lions were positive for CD out of 56 lions (87.5%) in a serosurvey on captive Asiatic lions from Western India. The evidence for current or past exposure to CDV was found in 90.7% of the population from a survey of the free dog population at Great Indian Bustard Sanctuary, Maharashtra, India (Vanak *et al.*, 2007). Srivastav and Nigam, (2010) reported that CD was a major threat to the Asiatic wild dog population in India.

Pawar *et al.* (2011) for the very first time reported the isolation of Indian CDV from B95a cells and sequence analysis of the N gene was done. To the best of our knowledge, the first report on sequencing and characterization of full-length F, P, and M genes of wild-type CDV from India was reported by Swati *et al.* (2015). They mainly focused on the cloning, sequencing, and phylogenetic analysis of an Indian CDV strain; compared against the reference CDVs available from GenBank and the commercial CDV vaccine strains.

Rajashekar *et al.* (2023) reported the incidence of CD was 19.04%. The maximum cases (45%) were found in dogs of 0-6 months of age. Males (75%) were affected more than females. Mongrels were most affected (55%), followed

by German Shepherds (15%), Labrador and Doberman (10% each), Pomeranians and crossbreeds (5%).

Host Range and Susceptibility

CDV is closely related to other morbilliviruses such as measles virus (MV) of human and nonhuman primates; rinderpest virus (RPV) of cattle, pigs, goats, sheep, buffalos, elands, giraffes, kudus, and warthogs and pestes des petite ruminants' virus (PPRV) of goats, sheep, gazelles and ibexes (Dungworth,1993 and Osterhaus *et al.*, 1995). CDV infects other mammals including species of the families Mustelidae (ferret, mink, skunk, weasel, badger, and marten), Procyonidae (raccoon, coati and kinkajou), Ailuridae (lesser and giant pandas), Ursidae (bear), Viverridae (fossa, mongoose, civet, linsang, binturong, and genet), Hyaenidae (hyena), and Felidae (cheetah, lion, jaguar, margay, tiger, cat, and ocelot) (Dungworth, 1993).

Structure

CDV is a single-stranded, negative-sense (3'-5'), non-segmented, lipoprotein-enveloped RNA virus with a diameter of about 150-300 nm. The genome of CDV is about 15.7 kb, which consists of six genes encoding six structural proteins (Murphy *et al.*, 1999). The organization of the major gene codes in the CDV genome is 3´-N-P-M-F-H- L-5´, each separated by UTRs (Griffin, 2001). Six genes encode six structural proteins; two glycoproteins (H and F protein), a single envelope-associated M protein, two transcriptase-associated proteins (P, L), and the nucleo-capsid protein (N) that encapsidates the viral RNA (Van Regenmortel *et al.*, 2000). The non-structural proteins (C) and (V) were produced by an alternative open reading frame in the P gene (Lamb and Kolakofsky, 2001). The schematic representation of the CDV genome is shown in **Fig. 1.** The hemagglutinin or "H" glycoprotein is responsible for the viral attachment to the host cell (Murphy *et al.*, 1999). The fusion (F) protein is a glycoprotein that is essential for mediating fusion between the viral particle and the host cell membrane. The F protein provides the virus with the necessary mechanism to move from one host cell to another (Murphy *et al.*, 1999). The matrix (M) protein is important for the entry of the virus into a susceptible cell and this protein plays a critical role in the assembly of new viral particles (Murphy *et al.*, 1999). The phospholipid (P) and the large (L) proteins form a functional polymerase complex and are responsible for the replication of viral RNA. The P protein also encodes for two non-structural proteins, C and V. The nucleocapsid (NP) protein is responsible for protecting the viral RNA (Murphy *et al.*, 1999).

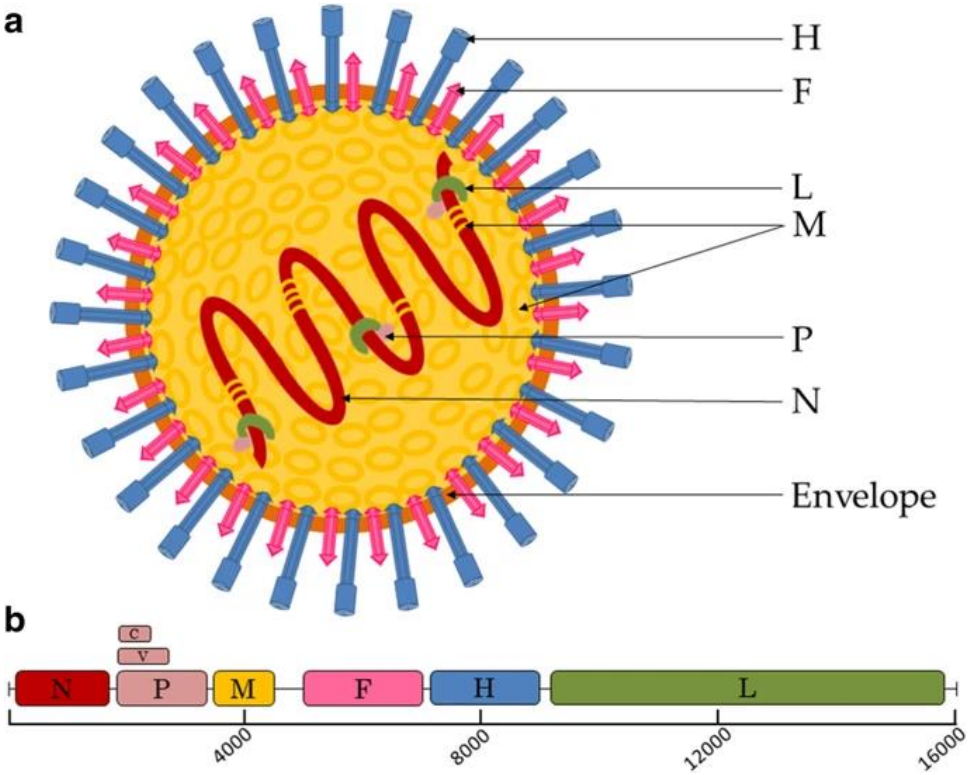

Fig. 1: Schematic representation of the ultrastructural morphology and genetic features of CDV

Virion Properties

CDV is extremely sensitive to UV radiation, heat desiccation (50-60°C), oxidizing agents, detergents, and lipid solvents. At room temperature the virus is short-lived, surviving between 20 minutes to 2 hours in tissues and exudates. Viruses can survive for several days at temperatures below zero if protected by organic material (Greene and Appel. 1984). The virus remains viable between pH 4.5-5, and it is susceptible to organic solvents like ether, chloroform, dilute (< 5%) formalin solution, phenol (0.75%), and quaternary ammonium disinfectant (0.3%). CDV in infected tissues has been shown to retain infectivity for days at 25°C and weeks at 2-4°C (Shen and Gorham, 1980). CDV can survive at -65ºC for at least 7 years (Greene & Appel 1998).

Transmission

The most common source of CDV infection is direct contact between susceptible dogs and infected dogs or wildlife. CDV is commonly transmitted by aerosol or droplet exposure originating from respiratory exudates; however, urine and other secretions also contain an infectious virus (Greene & Appel, 1998). Its incubation period ranges from about 1 week to 1 month. Canine distemper is highly contagious, and viral shedding may continue for 60–90 days after infection. (Appel 1987). Transplacental infection has been documented in domestic dogs. (Krakowka *et al.*, 1977).

Clinical Signs

A transient fever usually occurs 3–6 days after infection with canine distemper virus, and there may be leukopenia (characterized by lymphopenia) at this time; these clinical signs may go unnoticed or may be accompanied by anorexia. The fever subsides for several days. As the virus spreads to the respiratory and gastero intestinal system, clinical signs such as vomiting, diarrhea, fever, reduced appetite, respiratory signs such as mucopurulent oculonasal discharges, conjunctivitis, respiratory distress, and lethargy can be seen. Encephalomyelitis may occur in association with these signs, follow the systemic disease, or occur in the absence of systemic signs. Dogs surviving the acute phase may have hyperkeratosis of the footpads (*HARDPAD DISEASE*) and epithelium of the nasal planum, as well as enamel hypoplasia in incompletely erupted teeth. Typical neurologic signs include localized involuntary muscle twitching (myoclonus, chorea, flexor spasm, hyperkinesia) and seizures, including salivation and chewing movements of the jaw (Old Dog Encephalitis) (www.msdvetmanual.com).

Diagnosis

Tentative diagnosis is typically done through clinical signs and laboratory tests. Confirmatory diagnosis is done by RT-PCR, antibody detection tests, isolation and identification of organisms. Molecular diagnosis can be done using PCR. PCR is a highly sensitive, rapid, specific technique that can be used for molecular identification of viruses (Frisk *et al.*, 1999).

Prevention and Treatment

The best way to prevent CD is through Vaccination. The canine distemper vaccine is included in a combination vaccine (sometimes abbreviated DAPP, DA2PP). This vaccine is considered “core” and is recommended for all dogs. To help them build immunity, puppies need to undergo an initial series of vaccinations at 45-60 days and a second dose 1 month later, followed by a booster dose every year to maintain immunity as adults. Keep your dog away from other dogs when sick, including other dogs within your home. Avoid contact with known infected dogs and their premises. Keep your dog away from wildlife (www.avma.org).

There is no cure, and no antiviral drugs have been approved to combat the infection. Treatment usually consists of supportive care such as fluids to correct dehydration and medications to prevent secondary infections and control vomiting, diarrhea, and neurologic signs. (www.avma.org).

Contribution by Authors

Equal contribution. All authors declared that ‘written informed’ consent was obtained from the approved parties for

the publication of this article and accompanying images.

Conflict of Interests

There is no conflict of interest.

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