

Canine Parvovirus: Leading cause of fatality in pups

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ABSTRACT

Canine parvovirus is a highly contagious viral disease of wild and domestic canids which is caused by Canine parvovirus Type-2. The virus enters the host body through ingestion and replicates in the oropharynx from where it spreads to the other body's organ via the bloodstream. The enteric form of the disease occurs due to the replication of the virus in intestinal crypts, and it exhibits characteristic signs of acute hemorrhagic enteritis, foul-smelling bloody diarrhea, vomiting, dehydration, and ultimately leads to death. While cardiac form of the disease occurs due to scarring of the myocardium and is characterized by myocarditis and myocardial necrosis. Diagnosis of CPV is based on in-clinic assays and laboratory tests. Despite the provision of intensive care and rigorous supportive treatment, the survival rates of animals are still low. Control and prevention of disease is based on immunization via modified live vaccines but vaccine failure due to multiple reasons is also reported.

1. Introduction:

Canine parvovirus is an infectious disease of the members of Family Canidae (Dogs, wolves, coyotes, and foxes) which is responsible for hemorrhagic enteritis and myocarditis in dogs of all ages, but puppies less than 6 months old are particularly susceptible to the fatal outcomes of this disease [1]. Canine parvovirus-2 causes this disease, and it is thought to be a mutant variant of Feline panleukopenia virus (FPLV) due to its genomic similarity with it [2, 3]. Outbreaks are characterized by high morbidity and mortality (10% in adult dogs and 91% in puppies). The virus enters the body through ingestion of contaminated food or water and replicates at specific sites such as oropharynx and intestinal crypts [4, 5, 6]. Prodromal signs of this disease are inappetence and lethargy which further leads to bloody diarrhea, listlessness, fever and vomiting [7]. It not only affects the gastrointestinal tract but also results in systemic inflammatory responses [5]. Definitive diagnosis is made by DNA-based studies although other serological methods or in-clinic assays are also present, but they are less sensitive [2, 8]. Parvoviral enteritis has no specific treatment and only supportive treatment is given [8]. Eradication of this virus is nearly impossible because it also circulates in wild animals which constantly spread disease in naïve domestic population of dogs. Vaccines against CPV may be found alone or in conjugation with any other canine pathogen. But cases of vaccine failure have also been reported due to interfering maternal antibodies, a non-responding host, lack of annual revaccination, inappropriate degree of attenuation, insufficient time between vaccination and exposure, or concurrent disease ultimately leading to the occurrence of disease in vaccinated animals [9]. CPV-2 has no public health significance [10].

2. Etiology

Canine parvovirus (CPV) is an infectious viral disease caused by a small, icosahedral, non-enveloped, single-stranded negative sense DNA virus that belongs to the family *Parvoviridae*, sub-family *Parvovirinae* and genus *Parvovirus*. This is about 98% identical to the Feline panleukopenia virus (FPLV) and considered to be a mutant of it [2]. It has two non-structural proteins, i.e NS1, NS2 and three structural proteins, i.e VP1, VP2, and VP3 [3]. Canine parvovirus type-2 is antigenically unrelated to Canine parvovirus type-1 (Minute virus of canines) which may lead to pneumonia and early embryonic mortality in addition to other gastric or cardiac symptoms. CPV type-2 is further classified into three variants: CPV type-2a, CPV type-2b, CPV type-2c. They are mainly associated with enteric or cardiac disease [2]. The prevalence of these variants vary from country to country or within different regions of the same country.

3. Host Susceptibility

All the breeds, sexes and age groups of dogs and other wild or domestic canids are susceptible to CPV-2 infection, but puppies under 6 months lacking maternal-derived antibodies are particularly at risk [1]. It is the leading cause of mortality due to circulatory shock or myocarditis.

4. Pathogenesis

CPV affects epithelial cells of intestinal crypts, lymph nodes, thymus, and bone-marrow precursor cell through virus-induced destruction [4]. The virus gains entrance into the body through ingestion and replicates in the oropharynx after two days and spreads to other organs via bloodstream [5]. After 3-5 days viremia is evident in infected animals. It not only causes enteric disease but also systemic infection affecting multiple organs and organ systems. After reaching the intestinal mucosa, virus replicates and kills germinal epithelial cells of the intestinal crypts rendering their physiological function leading to enteric disturbances which leads to diarrhea, vomiting, anorexia and other symptoms related to digestion as shown in (Fig.1).

Destruction of myeloproliferative cells and lymphoid necrosis ultimately leads to lymphopenia. A chronic progressive form of the disease is cardiac form which leads to myocarditis. The virus is hardy and resistant in the environment with a very low infectious dose of few hundred TCID₅₀ (Tissue culture infectious dose) sufficient to cause disease and shed up to 10 billion TCID₅₀/g in feces of infected animal. Thus, transmission of virus in healthy animal occurs via direct or indirect route [6]. Direct transmission occurs via ingestion of fecal material of infected animal. While indirect transmission occurs via ingesting food or water contaminated with feces.

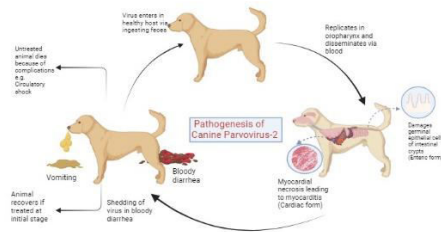


Fig. 1: Pathogenesis of Canine Parvovirus-2 (Retrieved from Bio-Render)

5. Clinical Manifestation

The severity of Canine parvovirus ranges from an asymptomatic form to acute or chronic form. There are two clinical forms of disease namely enteric form and cardiac form. Former is manifested by gastroenteritis while later is manifested by myocarditis leading to myocardial necrosis which is prevalent in young puppies lacking immunization by maternal antibodies. The hematological findings comprise thrombocytopenia and leucopenia. Acute hemorrhagic gastroenteritis, anorexia, vomiting, depression, dehydration, foul-smelling diarrhea are the cardinal signs of this disease. On general examination prolonged capillary refill time, tachycardia and low rectal temperature will be evident in late form of disease [7]. Animal recovering from acute gastroenteritis have good prognosis but complication due to severe dehydration, suppression of immune system and circulatory shock ultimately leads to death of animal despite intensive care and rigorous treatment.

6. Diagnosis

Canine parvovirus can often be confused with other causes of gastrointestinal disturbance e.g. non-infectious enteritis, so it is needed to be differentially diagnosed in initial stages of disease. In-clinic assays and laboratory tests are widely used for this purpose. In-clinic assays are cost-effective and easy to perform but they are less sensitive. Laboratory tests include serological methods (ELISA, HA, CFT), molecular techniques (PCR) and virus isolation. DNA based assays are the most sensitive [2, 8]. These lab tests are time consuming and expensive but more sensitive and better option for diagnosis.

7. Treatment

There is no specific treatment of canine parvovirus, and only symptomatic or supportive treatment can be carried out [2, 8]. Unconventional treatment includes use of Osetamivir which interferes with ability of virus to proliferate in intestinal crypts and reduce the bacterial colonization. Supportive therapy includes fluid therapy with balanced formula of electrolytes and water-soluble vitamins. Stomach protection agents, antibiotics, anti-emetics and Vitamin K are also administered to affected puppies.

8. Control and Prevention

Isolation of infected animals from healthy animals can control the spread of disease in animals that are housed at same place. Despite extensive

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vaccination, incidences of canine parvovirus and mortality may occur due to vaccination failure [9]. Major reasons behind vaccination failure include interference of maternal antibody titer, presence of non-respondents and other host or environment related factors. Antibodies and cell-mediated immune response are stimulated via modified live vaccine, but they have low immunogenicity, and it needs to be frequently administered. Inactivated vaccines are usually not used because they are unable to mount sufficient immune response. Thus, vaccinating the animals against CPV can reduce the chances of disease outbreak. Canine parvovirus is not a zoonotic disease and CPV-2 variants are not transmitted to human from infected animal [10].

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