

Canine Parvovirus Infection

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Introduction

Canine parvovirus (CPV) is an infectious ailment primarily targeting dogs. It exhibits a high level of contagiousness, disseminating among dogs through direct or indirect interactions. Vaccination offers preventive measures against this disease; however, in the absence of treatment, the fatality rate can soar to 91%. Typically, treatment necessitates hospitalization. Notably, canine parvovirus has the potential to infect other animals as well, encompassing species like wolves, cats, and skunks. Among felines, the susceptibility extends to panleukopenia, a distinct variant of the parvovirus.

Canine parvovirus exists in two types known as CPV1 and CPV2. CPV2 is responsible for the most severe form of the disease affecting both domestic dogs and wild canines. Among the variants of CPV2, CPV-2a and CPV-2b were identified in 1979 and 1984 respectively. These two strains are believed to cause most cases of canine parvovirus, replacing the original strains. Another variant, CPV-2c, a Glu-426 mutant, has been found in Italy, Vietnam, and Spain. While the antigenic structure of CPV-2a and CPV-2b is similar to the original CPV2, CPV-2c displays a distinct antigenicity pattern. Initial concerns about the effectiveness of dog vaccination against CPV-2c were later addressed through research, demonstrating that the current CPV-2b-based vaccine provides sufficient protection against CPV-2c.

Severe cases of infection can lead to canine death within 48 to 72 hours without treatment, while less severe cases have a mortality rate of approximately 10%. Certain breeds like Rottweilers, Dobermans, Pit Bulls, and other black and tan dogs might be more susceptible to CPV2. Alongside age and breed, factors such as

environmental stress, exposure to viruses, parasites, and canine coronaviruses contribute to a dog's risk of severe illness. The primary cause of death in dogs infected with parvovirus is dehydration or secondary infection, rather than the virus itself.

CPV2 variants are characterized by their surface protein (VP capsid) properties, which somewhat correspond with the virus genome's phylogenetic relationships. CPV2 is a non-enveloped, single-stranded DNA virus from the Parvoviridae family, with a diameter of only 20 to 26 nanometers. Its genome is around 5000 nucleotides long, and it displays icosahedral symmetry. CPV2 continues to evolve, adapting to host resistance and enhancing binding to its receptor, the canine transferrin receptor. It has a higher mutation rate compared to RNA viruses like influenza A.

While CPV2 predominantly affects dogs, wolves, and other canines, CPV2a and CPV2b have been found in a small portion of symptomatic cats. In the past, it was believed that CPV2 could not cross species, but studies in Vietnam have indicated minor immune system changes and natural transmission adaptations in felines. Notably, analysis of feline parvovirus (FPV) isolates revealed that a significant portion of isolates from Vietnam and Taiwan were canine parvovirus rather than feline panleukopenia virus (FPLV). CPV2 can be more easily transmitted to cats than dogs and exhibits a faster mutation rate in this species.

Enteric form

Dogs contract CPV2 through oral exposure to feces, contaminated soil, or contaminated objects. Once the virus is ingested, it replicates in the lymphoid tissue of the pharynx and then enters

the bloodstream. The virus subsequently targets rapidly dividing cells, with a preference for tumors, intestinal crypts, and bone marrow. This leads to lymphopenia in lymph nodes and the necrosis and damage of intestinal crypts. Concurrently, anaerobic bacteria that normally reside in the intestinal tract can enter the bloodstream through a process known as translocation, resulting in bacteremia and sepsis. Notable pathogens involved include *Clostridium*, *Campylobacter*, and *Salmonella*. This scenario can give rise to systemic inflammatory response syndrome (SIRS), which carries potential complications such as blood hypercoagulation, endotoxemia, and acute respiratory distress syndrome (ARDS). Myocarditis as a result of sepsis has also been documented.

Additionally, dogs affected by CPV are susceptible to intussusception, a condition where one segment of the intestine becomes invaginated within another. In the days following infection, the virus is shed in the feces for up to three weeks, and infected dogs may remain carriers of the virus, shedding it regularly. In cases where the host is afflicted by other gastrointestinal diseases, the prognosis for CPV infection is typically grim, often leading to fatality.

Cardiac form

This particular variant is infrequent and primarily affects puppies that contract the disease either within the womb before 8 weeks of age or shortly after birth. This variation of the disease targets the heart muscle, often leading to swift puppy mortality either immediately or following brief episodes of labored breathing attributed to pulmonary edema. At the microscopic level, the myocardium displays multiple areas of necrosis accompanied by infiltration of mononuclear cells. In surviving puppies, an excessive formation of tissue (fibrosis) is usually evident. Muscle fibers serve as the site for intracellular replication of the virus. While symptoms of the intestinal form may or may not be present, this variant has become scarce due to widespread dog vaccination.

Less frequently, the virus can infiltrate the uterus, potentially causing harm and attacking various non-cardiac tissues such as the brain, liver, lungs, kidneys, and adrenal cortex. This results in replication, infection, and damage. The inner linings of blood vessels also experience severe damage, leading to hemorrhaging within the affected area.

Fetal infection emerges when a developing fetus becomes infected with CPV2 virus. While adults tend to develop immunity, the disease might manifest with minimal or no symptoms. The transmission of the virus to unborn offspring can induce complications. In mild to moderate cases, puppies might be born with conditions like cerebellar hypoplasia, which leads to underdeveloped brain structures.

Clinical Symptoms

The onset of disease symptoms usually occurs between three to ten days after exposure. These symptoms may encompass lethargy, vomiting, fever, and diarrhea, often characterized by the presence of blood. Lethargy often stands out as the initial indicator of canine parvovirus (CPV) infection. Subsequent symptoms entail weight loss, decreased appetite, and the progression from diarrhea to vomiting. The combined effects of diarrhea and vomiting can lead to dehydration, which in turn disrupts the dog's electrolyte balance. The weakened state of the body leaves it susceptible to secondary ailments.

The damage to the intestinal lining persists even as the infection advances, allowing blood and protein to enter the intestines. This results in both blood deficiency and protein loss. Moreover, endotoxins seep into the bloodstream, leading to a condition known as endotoxemia. A distinct odor is often noticeable in infected dogs. The reduction in white blood cells further contributes to the overall debility of the dog. Any or all of these factors can contribute to shock and even fatality. Puppies, in particular, have a poor prognosis and survival rate when afflicted with CPV.

Diagnosis

The diagnosis of canine parvovirus (CPV) infection involves various methods, including the detection of CPV2 in fecal samples using techniques such as ELISA, hemagglutination tests, or electron microscopy. Polymerase chain reaction (PCR) is also employed to identify CPV2, particularly in cases where virus shedding in the stool might be low and not detectable by ELISA. While ELISA can be used to detect virus shedding, PCR is more sensitive and can detect infections with lower viral loads.

Clinically, CPV infection can be mistaken for other enteric infections or coronaviruses, as they share similar symptoms. However, the severity of CPV infection sets it apart, characterized by symptoms like diarrhea, reduced white blood cell count, and necrosis of the

intestinal lining. These signs are particularly notable in unvaccinated dogs. On the other hand, diagnosing heart disease is usually straightforward due to the evident symptoms it presents.

Treatment

The survival outcome of canine parvovirus (CPV) infection hinges on several factors, including the promptness of diagnosis, the dog's age, and the aggressiveness of treatment. Presently, there is no universally agreed-upon clinical protocol, but the established standard of care involves supportive measures that necessitate extensive hospitalization. This is due to the risk of severe dehydration and damage to the gastrointestinal tract and bones.

When CPV is suspected, it is crucial to conduct CPV testing promptly to initiate treatment at an early stage, which can significantly enhance survival rates once the disease is confirmed. The optimal treatment regimen encompasses intravenous (IV) administration of crystalloid fluids and/or colloids, along with antiemetic drugs like maropitant, metoclopramide, dolasetron, ondansetron, and prochlorperazine. Broad-spectrum antibiotics such as cefazolin/enrofloxacin, ampicillin/penicillin/enrofloxacin, metronidazole, timentin, or enrofloxacin are also administered. Intravenous fluids are administered, and anti-nausea medications and antibiotics can be given subcutaneously, intramuscularly, or intravenously. These fluids typically contain electrolytes, B vitamins, dextrose, and potassium chloride.

Blood transfusions from CPV-recovered donor dogs are occasionally used to boost immunity in affected dogs. Some veterinarians employ methods like holding dogs in position or offering them ice cream, although the efficacy of such practices is not controlled. Additionally, infusions of fresh frozen plasma and human albumin can aid in correcting protein deficiencies and promoting tissue regeneration in severe cases. However, the use of colloids like hydroxyethyl starch, which impacts colloid osmotic pressure, remains controversial in dogs prone to future complications.

Once the dog starts to manage fluids independently, they are gradually weaned off IV fluids and introduced to solid foods gradually. The duration of oral antibiotics depends on the white blood cell count and the patient's immune response to secondary infections. If IV fluids are initiated promptly upon symptom appearance and a CPV

test confirms the diagnosis, mild symptoms may improve within two to three days. In more severe cases, recovery may take from five days to two weeks depending on the treatment approach. However, even with hospitalization, there are no guarantees of a complete recovery and survival.

Prevention

Prevention is the key to ensuring the health of your puppy, given the high contagiousness of this disease. Proper vaccination protocols should be initiated around 7-8 weeks of age and continued at intervals of 3-4 weeks until the puppy reaches at least 16 weeks of age. It's important to note that pregnant individuals should avoid vaccination, as it can pose risks to both the developing baby and the mother's health. The resilience of this disease is noteworthy; it can survive for up to a year in various organic materials like feces and soil. It can withstand a range of temperatures, both low and high. The sole household disinfectant effective against the virus is bleach. A solution of bleach diluted at a ratio of 1:10 can be employed for disinfection to effectively combat and eliminate parvovirus.

Puppies typically receive a series of vaccinations, starting as soon as maternal immunity wanes and continuing until the passive immunity is completely diminished. Older puppies, aged 16 weeks or more, undergo three vaccinations spaced 3 to 4 weeks apart. The duration of immunity conferred by CPV2 vaccines has been extensively studied by major vaccine manufacturers in the United States, indicating a minimum of three years after the initial puppy series and a booster one year later.

A dog that successfully recovers from CPV2 remains contagious for around three weeks, with a possibility of extending to six weeks. The ongoing risk of infection primarily arises from the fecal contamination of the environment due to the virus's remarkable ability to endure for several months in the surroundings. It's advisable to inform neighbors and household members with dogs about infected animals, allowing them to ensure that their pets are properly vaccinated or tested for immunity. A modified live vaccine can offer protection within 3 to 5 days; during this time, the infected individual should be kept in isolation until other animals are adequately protected.