Canine Diseases

Canine Parvovirosis

Canine parvovirosis is a highly contagious and relatively common cause of acute, infectious GI illness in young dogs caused by a relatively recently evolved virus, canine parvovirus 2 (CPV-2) which first emerged in the late 1970s. Although its exact origin is unknown, it is believed to have arisen from feline panleukopenia virus or a related parvovirus of nondomestic animals. The virus showed relatively rapid genetic evolution and within only a few years the first antigenic variants, CPV-2a and CPV-2b, appeared. These variants completely replaced the original CPV-2 in the field and spread worldwide. In 2000, a new variant, referred to as CPV-2c, was first identified in Europe and this variant has now also been identified in many other countries.

Etiology and Pathophysiology

CPV-2 is a nonenveloped, single-stranded DNA virus, resistant to many common detergents and disinfectants. Infectious CPV-2 can persist indoors at room temperature for a few weeks; outdoors, if protected from sunlight and desiccation, it persists for many months.

Young (6 wk to 6 mo), unvaccinated or incompletely vaccinated dogs are most susceptible. Rottweilers, Doberman Pinschers, American Pit Bull Terriers, English Springer Spaniels and German Shepherd dogs have been described to be at increased risk of disease. Assuming sufficient colostrum ingestion, puppies born to a dam with CPV-2 antibodies are protected from infection for the first few weeks of life; however, susceptibility to infection increases as maternally acquired antibody wanes. Stress (eg, from weaning, overcrowding, malnutrition, etc), concurrent intestinal parasitism, or enteric pathogen infection (eg, *Clostridium spp*, *Campylobacter spp*, *Salmonella spp*, *Giardia spp*, coronavirus) have been associated with more severe clinical illness.

Virus is shed in the feces of infected dogs within 4–5 days of exposure (often before clinical signs develop), throughout the period of illness, and for ~10 days after clinical recovery. Infection is acquired directly through contact with virus-containing feces or indirectly through contact with virus-contaminated fomites (eg, environment, personnel, equipment). Viral replication occurs initially in the lymphoid tissue of the oropharynx, with systemic illness resulting for subsequent hematogenous dissemination. CPV-2 preferentially infects and destroys rapidly dividing cells of the small intestinal crypt epithelium, lymphopoietic tissue, and bone marrow. Destruction of the intestinal crypt epithelium results in epithelial necrosis, villous atrophy, impaired absorptive capacity, and disrupted gut barrier function with the potential for bacterial translocation and bacteremia.

Lymphopenia and neutropenia develop secondary to destruction of hematopoietic progenitor cells in the bone marrow and lymphopoietic tissues (eg, thymus, lymph nodes, etc) and are further exacerbated by an increased systemic demand for leukocytes. Infection in utero or in pups <8wk old or born to unvaccinated dams without naturally occurring antibodies can result in myocardial infection, necrosis, and myocarditis. Myocarditis, presenting as acute cardiopulmonary failure or delayed, progressive cardiac failure, can occur with or without signs of enteritis. However, CPV-2 myocarditis is infrequent because most bitches have CPV-2 antibodies from immunization or natural exposure.

Clinical and Pathological Findings

Clinical signs of parvoviral enteritis generally develop within 3–7 days of infection. Initial clinical signs may be nonspecific (eg, lethargy, anorexia, fever) with progression to vomiting and hemorrhagic small-bowel diarrhea within 24–48 hr. Physical examination findings can include depression, fever, dehydration, and intestinal loops that are dilated and fluid filled. Abdominal pain warrants further investigation to rule out the potential complication of intussusception. Severely affected animals may present collapsed with prolonged capillary refill time, poor pulse quality, tachycardia, and hypothermia—signs potentially consistent with septic shock. Although CPV-2-associated leukoencephalomalacia has been reported, CNS signs are more commonly attributable to hypoglycemia, sepsis, or acid-base and electrolyte abnormalities. Inapparent or subclinical infection is common.

Gross necropsy lesions can include a thickened and discolored intestinal wall; watery, mucoid, or hemorrhagic intestinal contents; edema and congestion of abdominal and thoracic lymph nodes; thymic atrophy; and, in the case of CPV-2 myocarditis, pale streaks in the myocardium. Histologically, intestinal lesions are characterized by multifocal necrosis of the crypt epithelium, loss of crypt architecture, and villous blunting and sloughing. Depletion of lymphoid tissue and cortical lymphocytes (Peyer's patches, peripheral lymph nodes, mesenteric lymph nodes, thymus, spleen) and bone marrow hypoplasia are also observed. Pulmonary edema, alveolitis, and bacterial colonization of the lungs and liver may be seen in dogs that died of complicating acute respiratory distress syndrome, systemic inflammatory response syndrome, endotoxemia, or septicemia.

Diagnosis

CPV-2 enteritis should be suspected in any young, unvaccinated, or incompletely vaccinated dog with relevant clinical signs. Over the course of the illness, most dogs develop a moderate to severe leukopenia characterized by lymphopenia and neutropenia. Leukopenia, lymphopenia, and the absence of a band neutrophil response within 24 hr of initiating treatment has been associated with a poor prognosis. Prerenal azotemia, hypoalbuminemia (GI protein loss), hyponatremia, hypokalemia, hypochloremia, and hypoglycemia (inadequate glycogen stores in young puppies, sepsis), and increased liver enzyme activities may be noted on serum biochemical profile. Commercial ELISAs for detection of antigen in feces are widely available. Most clinically ill dogs shed large quantities of virus in the feces. However, false-negative results can occur early in the course of the disease (before peak viral shedding) and after the rapid decline in viral shedding that tends to occur within 10–12 days of infection. False-positive results can occur with 4–10 days of vaccination with modified live CPV-2 vaccine. Alternative methods of detecting CPV-2 antigen in feces include PCR testing, electron microscopy, and virus isolation. Serodiagnosis of CPV-2 infection requires demonstration of a 4-fold increase in serum IgG titer over a 14-day period or detection of IgM antibodies in the absence of recent (within 4 wk) vaccination.