**Etiology and Pathophysiology**

Canine parvovirus (CPV) is a highly contagious and relatively common cause of acute, infectious GI illness in young dogs. Although its exact origin is unknown, it is believed to have arisen from feline panleukopenia virus or a related parvovirus of nondomestic animals. It is a nonenveloped, single-stranded DNA virus, resistant to many common detergents and disinfectants. Infectious CPV can persist indoors at room temperature for up to 2 mo; outdoors, if protected from sunlight and desiccation, it persists for up to 5 mo. In North America, clinical disease is largely attributed to CPV-2b; however, infection with a new and equally virulent strain, CPV-2c, is increasingly common.

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 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. Among dogs >6 mo old, intact male dogs are more likely than intact female dogs to develop CPV enteritis.

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 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 antibodies from immunization or natural exposure.

**Clinical 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-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.

***Lesions***

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 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 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 ELISA 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 vaccine. Alternative methods of detecting CPV antigen in feces include PCR testing, electron microscopy, and virus isolation. Serodiagnosis of CPV 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.

**Treatment and Prognosis**

The main goals of treatment for CPV enteritis include restoration of fluid, electrolyte, and metabolic abnormalities and prevention of secondary bacterial infection. In the absence of significant vomiting, oral electrolyte solutions can be offered. Administration SC of an isotonic balanced electrolyte solution may be sufficient to correct mild fluid deficits (<5%) but is insufficient for dogs with moderate to severe dehydration. Most dogs will benefit from IV fluid therapy with a balanced electrolyte solution. Correcting dehydration, replacing ongoing fluid losses, and providing maintenance fluid needs is essential for effective treatment. Dogs must be monitored for development of hypokalemia and hypoglycemia. If electrolytes and serum blood glucose concentration cannot be routinely monitored, empirical supplementation of IV fluids with potassium (potassium chloride 20–40 mEq/L) and dextrose (2.5–5%) is appropriate.

If GI protein loss is severe (albumin <20 g/L, total protein <40 g/L, evidence of peripheral edema, ascites, pleural effusion, etc), colloid therapy should be considered. Nonprotein colloids (eg, pentastarch, hetastarch) can be administered in 5 mL/kg boluses (maximum of 20 mL/kg) over no less than 15 min. The remainder of the maximal 20 mL/kg dose can be administered as a constant-rate infusion over 24 hr and the volume of crystalloids administered decreased by 40–60%. Alternatively, transfusion of fresh frozen plasma may partially replace serum albumin while providing serum protease inhibitors to counter the systemic inflammatory response. There is no evidence to support the use of serum from dogs recovered from CPV-enteritis (convalescent or hyperimmune serum) as a means of passive immunization.

Antibiotics are indicated because of the risk of bacterial translocation across the disrupted intestinal epithelium and the like-lihood of concurrent neutropenia. A β-lactam antibiotic (eg, ampicillin or cefazolin [22 mg/kg, IV, tid]) will provide appropriate gram-positive and anaerobic coverage. For severe clinical signs and/or marked neutropenia, additional gram-negative coverage (eg, enrofloxacin [5 mg/kg, IM or IV, sid] or gentamicin [6 mg/kg, IV, sid]) is indicated. Aminoglycoside antibiotics must not be administered until dehydration has been corrected and fluid therapy established. Enrofloxacin has been associated with articular cartilage damage in rapidly growing dogs 2–8 mo old and should be discontinued if joint pain or swelling develops.

Antiemetic therapy is indicated if vomiting is protracted, perpetuates dehydration and electrolyte abnormalities, or limits oral administration of medications and nutritional support. α-Adrenergic antagonists (eg, prochlorperazine, 0.1–0.5 mg/kg, SC, tid) can worsen hypotension in hypovolemic animals, while prokinetic agents (eg, metoclopramide, 0.3 mg/kg, PO or SC, tid or 1–2 mg/kg/day constant-rate infusion) may increase the risk of intussusception; use of either agent should be restricted to rehydrated and appropriately monitored animals. The safety and efficacy of newer antiemetic agents such as ondansetron (0.1–0.2 mg/kg, given slowly IV, bid-qid) and maropitant (1 mg/kg, SC, sid for 5 days) have not been evaluated in CPV enteritis. Vomiting may persist despite antiemetic administration. Antidiarrheals are not recommended because retention of intestinal contents within a compromised gut increases the risk of bacterial translocation and systemic complications.

Previous recommendations for nutritional management of CPV enteritis included withholding food and water until cessation of vomiting, but recent evidence suggests early enteral nutrition is associated with earlier clinical improvement, weight gain, and improved gut barrier function. For anorectic dogs, placement of a nasoesophageal or nasogastric tube and bolus or feeding a prepared liquid diet (eg, Clinicare®, or dilute, blended canned diet) should be instituted within 12 hr of admission to hospital. Once vomiting has subsided for 12–24 hr, gradual reintroduction of water and a bland, low-fat, easily digestible commercial or homemade (eg, boiled chicken or low-fat cottage cheese and rice) diet is recommended. Partial or total parenteral nutrition is reserved for dogs with anorexia >3 days that are intolerant of enteral feeding.

Initial evidence suggests administration of recombinant feline interferon-ω (2.5 U/kg, IV, sid for 3 consecutive days) lessens clinical signs and mortality due to CPV enteritis. Treatment with the antiviral agent oseltamivir (2 mg/kg, PO, bid for 5 days) can be considered; however, reports to support its efficacy are lacking. The potential for induction of or selection for resistance to influenza virus have led some to recommend that oseltamivir therapy be avoided in CPV enteritis. Adverse effects potentially attributable to oseltamivir after 3 days of therapy include lethargy, abdominal pain, gastric dilation, diarrhea, and restlessness. Other adjunctive treatments such as recombinant human granulocyte colony-stimulating factor and recombinant bactericidal/permeability-increasing protein have not been beneficial.

Intussusception, bacterial colonization of IV catheters, thrombosis, urinary tract infection, septicemia, endotoxemia, acute respiratory distress syndrome, and sudden death are potential complications of CPV enteritis. Most puppies that survive the first 3–4 days of illness make a full recovery, usually within 1 wk. With appropriate supportive care, 68–92% of dogs with CPV enteritis will survive. Dogs that recover develop longterm, possibly life-long immunity.

**Prevention and Control**

To limit environmental contamination and spread to other susceptible animals, dogs with confirmed or suspected CPV enteritis must be handled with strict isolation procedures (eg, isolation housing, gowning and gloving of personnel, frequent and thorough cleaning, etc). All surfaces should be cleaned with a solution of dilute bleach (1:30), peroxygen, or an accelerated hydrogen peroxide disinfectant. The same solutions may be used as footbaths to disinfect footwear.

To prevent and control CPV, vaccination with a modified live vaccine is recommended at 6–8, 10–12, and 14–16 wk of age, followed by a booster administered 1 yr later and then every 3 yr. Because of potential damage by CPV to myocardial or cerebellar cells, inactivated rather than modified live vaccines are indicated in pregnant dogs or colostrum-deprived puppies vaccinated before 6–8 wk of age. The presence of maternally acquired CPV antibodies may interfere with the effectiveness of vaccination in puppies <8–10 wk old. Current modified live CPV vaccines are sufficiently immunogenic to protect puppies from infection in the presence of low levels of interfering maternal antibody. At least 2 commercial vaccines provide protection against the CPV-2c variant circulating in the USA.

A new puppy should be introduced into the home of a dog recently diagnosed with CPV enteritis no sooner than 1 mo after clinical signs have resolved. Only fully vaccinated puppies (6, 8, and 12 wk vaccines) should be considered. Introduction of an incompletely vaccinated adult dog should be handled similarly. Booster vaccination of in-contact healthy dogs that are up-to-date on parvovirus vaccination is reasonable but potentially unnecessary given the extended duration of immunity to CPV.

* *The Merck Veterinary Manual: Canine Parvo Virus*