Yekaterina Buriko, DVM, and Cynthia M. Otto, DVM, PhD, Diplomate ACVECC, University of Pennsylvania

Parvoviral Enteritis

Profile

Definition

Worldwide disease caused by CPV-2.

Signalment

Any unvaccinated dog is at risk for acquiring CPV, but illness is more prevalent in the following groups:

- Puppies between 6 weeks and 6 months of age
- Certain breeds: Rottweiler, Doberman pinscher, American pit bull terrier, German shepherd, Labrador retriever
- Sexually intact males older than 6 months of age: twice as likely as sexually intact females to acquire CPV[1]

Causes

- Parvovirus—small, nonenveloped, single-stranded DNA virus.
- Two virulent strains: CPV-2a and CPV-2b. Most cases in the United States are caused by CPV-2b.[2]

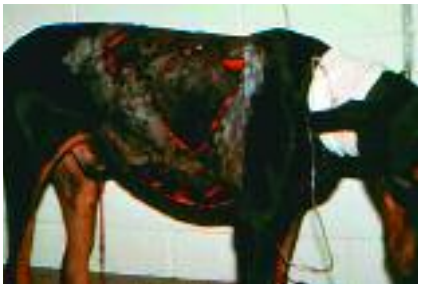
Risk Factors

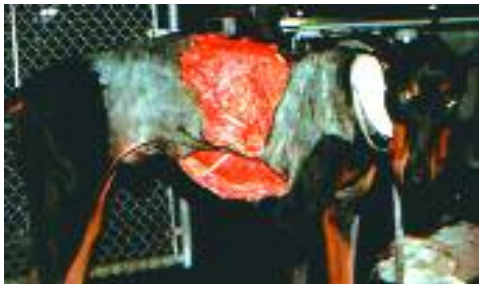
- Lack of protective immunity
- Unsanitary or overcrowded environments
- Endoparasitism
- Season: higher incidence between July and September
- Recent weaning—enterocytes have a higher mitotic index because of diet changes and changes in bacterial flora[1]

Pathophysiology

- Fecal-oral route of transmission.
- 2 days after ingestion, virus replicates in the oropharynx and local lymphoid tissue.

CPV = canine parvovirus; DNA = deoxyribonucleic acid





Infection and sloughing of the skin secondary to subcutaneous fluid administration in a dehydrated and immunocompromised dog with parvoviral infection. This is a rare but serious complication.

- Viremia peaks by 3 to 5 days after infection.[2]
- CPV preferentially affects rapidly dividing cells:
  - Lymphoid tissues
  - Intestinal epithelium
    - Virus reaches the intestinal mucosa via the bloodstream and replicates in the germinal epithelium of the intestinal crypts.
    - Damage to the intestinal crypts leads to villous atrophy, collapse of the intestinal epithelium, and loss of absorptive capacity.[2]
  - Bone marrow
    - Lymphoid necrosis and destruction of myeloproliferative cells in the bone marrow
    - Resultant lymphopenia and panleukopenia in severe cases
  - Myocardium, if infection occurs in utero or within the first 2 weeks of life; secondary liquefactive necrosis of the cerebral white matter[1]
- Fecal shedding starts approximately 3 days after infection, peaks on days 4 to 7, and continues for up to 2 weeks after infection.[3]

Signs

History

Anorexia, lethargy, vomiting, diarrhea (typically small bowel and hemorrhagic)

Physical examination

- Progressive dehydration, possibly hypovolemic shock: tachycardia, poor pulse quality, pale mucous membranes, prolonged capillary refill time, cool extremities, elevated or decreased body temperature. Abdominal pain is frequently elicited on palpation. Intussusception may be palpated in the form of an abdominal mass.[1]

Pain Index

Abdominal pain due to acute gastroenteritis or intussusception can range from mild to severe, depending on severity of infection.

Diagnosis

Definitive Diagnosis

Relies on demonstration of viral antigen in feces

continues

- ELISA
  - Convenient, fast, in-house test
  - Detects CPV antigen in feces
  - False-negative findings may result from serum-neutralizing antibodies in bloody diarrhea that bind CPV antigen
  - False-positive findings may result from vaccine virus shedding 3 to 10 days after vaccination with a modified live vaccine[1]
- PCR (feces)
  - Requires few particles of CPV—enhanced sensitivity
  - Highly specific and can differentiate wild-type infection from viral shedding secondary to vaccination
  - Not routinely available[4]
- Other methods of antigen detection—electron microscopy, viral isolation, stool hemagglutination, latex agglutination, counterimmunoelectrophoresis—require a diagnostic laboratory; not practical.
- Serology
  - Antibodies are detectable when clinical signs occur; titers rise rapidly and are high for years.
  - Anti-CPV IgM and typical clinical signs are diagnostic.
  - Lack of anti-CPV antibodies in dog with clinical signs of gastroenteritis can rule out CPV infection.
  - Has limited diagnostic value—large portion of healthy unvaccinated dogs are seropositive secondary to subclinical infections.[4]

Necropsy with Histopathology

Gross

- Jejunum and ileum are frequently affected
- Segmental hemorrhage and congestion
- Mesenteric lymphadenomegaly with cortical hemorrhage and necrosis; atrophy of thymus may be present.[4]

Microscopic

- Intestinal crypt necrosis
- Dilation of the crypts with necrotic debris; villous collapse and destruction
- Evidence of regeneration usually present[4]

Differential Diagnosis for Vomiting and Diarrhea

- Infectious causes (bacterial, viral, parasitic, protozoal)
- Dietary indiscretion
- Toxin ingestion
- Foreign body/intussusception
- Pancreatitis
- Metabolic (liver, renal disease, Addison's disease)
- Peritonitis/inflammation/infection outside of gastrointestinal tract

Laboratory Findings/Imaging

Dogs with CPV infection exhibit hypercoagulability, measured with thromboelastography.[5]

Complete blood count

- Frequent leukopenia due to lymphopenia and/or neutropenia with WBC counts frequently less than 2000 cells/µl
- Anemia—observed in patients with concurrent parasitic infections

Serum chemistry abnormalities

Electrolyte disturbances (most frequently hypokalemia); prerenal azotemia; hypoalbuminemia; elevations in ALT, ALP, and bilirubin levels

Abdominal radiographs

- Gas- and fluid-distended loops of small bowel due to enteritis
- Obstructive pattern if intussusception has occurred

Treatment

Inpatient or Outpatient

Because most dogs with CPV suffer from moderate to severe dehydration and have ongoing fluid losses, inpatient care is recommended. Outpatient management may be considered if the patient is mildly affected; frequent rechecks are essential to determine progression of disease.[1]

Activity

No activity restriction is necessary after recovery from CPV infection.

Client Education

- Other immature or poorly vaccinated dogs in the household are at risk for developing CPV infection and should be evaluated by a veterinarian. All objects with which the infected dog has come into contact should be disinfected with 1:32 dilution of bleach.
- CPV is ubiquitous and survives in the environment for months; the owners should refrain from obtaining a puppy for at least 6 months.
- Dogs with CPV infection shed large amounts of virus for up to 2 weeks after resolution of infection; the recovering dog should be isolated for 2 weeks.

Surgery

Emergency surgery is indicated in the event of intussusception.

Medications: Drugs/Fluids

- Intravenous fluid administration is the mainstay of therapy. A balanced crystalloid solution should be used initially. If the patient is exhibiting signs of shock, quick replacement of deficits is imperative. Various patients will need different amounts of fluids to restore perfusion, assessed by heart rate; mucous membrane color; capillary refill time; and blood pressure. Starting with a quarter to a third of the shock dose of fluids (90 ml/kg per hour) over 15 to 20 minutes is appropriate, followed by adjustments as needed. Intraosseous catheter may be used if circulatory collapse has occurred and intravenous access cannot be obtained. If the patient is dehydrated but

ALP = alkaline phosphatase; ALT = alanine aminotransferase; CPV = canine parvovirus; ELISA = enzyme-linked immunosorbent assay; H_2 = histamine$_2$; IgM = immunoglobulin M; PCR = polymerase chain reaction; WBC = white blood cell

is not in shock, deficits should be replaced over 6 to 24 hours

Deficit (L) = % dehydration × body weight (kg).

Fluid rate per hour may be calculated as follows:

Deficit/6-24 h + maintenance (2-4 ml/h) + ongoing losses/h.

Potassium and glucose supplementation is often indicated.

- Colloid solutions at 20 ml/kg per day should be considered if albumin level is less than 2 g/dl, total solids less than 4 g/dl, or evidence of peripheral edema or third spacing is present.
- Antibiotic therapy is indicated in dogs with disruption of the intestinal mucosal barrier or severe neutropenia. Intravenous broad-spectrum antibiotics are used, such as combination of ampicillin (22 mg/kg Q 8 H) and 1 of the following: enrofloxacin, amikacin, or gentamicin (15 mg/kg Q 24 H) (see **Contraindications/Precautions**). Alternatively, a single agent, such as cefoxitin (15 to 30 mg/kg Q 6 H) may be used.
- Antiemetic therapy is indicated if persistent vomiting occurs. Metoclopramide (1 to 2 mg/kg Q 24 H) may be used. If that is ineffective, a phenothiazine derivative (dolasetron or ondansetron) should be considered.[3]
- Fresh frozen plasma (10 to 20 ml/kg Q 8 to 12 H) should be administered in patients that have established coagulopathies and exhibit signs of disseminated intravascular coagulation. Plasma has also been recommended to provide immunoglobulins and serum protease inhibitors.[6]
- Treatment of concurrent or predisposing conditions (parasites) optimizes recovery time.
- Pain management with butorphanol (0.2 to 0.4 mg/kg IV Q 2-4 H) or buprenorphine (0.01 to 0.02 mg/kg IV Q 4-6 H).
- H_2 antagonists (such as famotidine 0.5 mg/kg IV Q 12-24 H) to minimize reflux esophagitis.

Contraindications/Precautions/Interactions

- Aminoglycosides should not be administered to dehydrated patients. If these drugs are used, urine sediment should be monitored daily for presence of tubular casts.
- Enrofloxacin may cause cartilage abnormalities in growing dogs.
- Phenothiazine derivatives: may cause vasodilation and hypotension and should not be used in dehydrated patients.
- Subcutaneous fluids in dehydrated and immunocompromised dogs may result in subcutaneous infections and sloughing of the skin.

Alternative Therapy

- Feline interferon-β: reduces mortality in experimental CPV infections; however, clinical use is limited.[7]
- Oseltamivir (Tamiflu): advocated in treatment of CPV; controlled studies are necessary to evaluate its efficacy.

Nutritional Aspects

- Stop all oral intake until vomiting has stopped for 24 hours.
- Institute early enteral nutrition
  - Essential to healing of the gastrointestinal tract
  - Could be achieved through oral feedings or feeding-tube placement if indicated
- Parenteral nutrition may be necessary for severe CPV infection.

Follow-up

Patient Monitoring

Physical measures and blood pressure should be monitored at least twice daily in hospitalized patients. Packed cell volume, total solids, blood glucose, electrolytes, acid-base status, and weight should be monitored at least daily.

Prevention

Modified live-virus vaccine is the vaccine of choice for preventing CPV infection. The biggest cause of vaccine failure is maternal antibody interference.[1]

Complications

Aspiration pneumonia due to vomiting, sepsis, and systemic inflammatory response syndrome can be observed.

Course

Average recovery time is 6 days.

At-Home Treatment

Once patient has recovered, vaccination for other infectious diseases is essential.

In General

Relative Cost

Outpatient management costs are lower than inpatient costs ($-$$). With severe CPV infection, inpatient care costs are high ($$$$-$$$$$).

Cost Key

$ = < $100	$$$$ = $500-1000
$$ = $100-250	$$$$$ = > $1000
$$$ = $250-500	

Prognosis

Prognosis for survival is good if aggressive supportive care is instituted early in disease.

See Aids & Resources, back page, for references, contacts, and appendices.