Update on Canine Parvovirus

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KEYWORDS

• Parvovirus • Enteritis • Treatment • Outpatient therapy

KEY POINTS

- Proper vaccination is the most important strategy for the prevention of canine parvovirus.
- The administration of intravenous fluids to maintain euvolemia and hydration is critical in the treatment of puppies with canine parvovirus.
- Enteral nutrition, gastrointestinal support medications, analgesics, and antibiotics are important components of supportive care.
- Placement of a multilumen central venous catheter will allow for the administration of multiple intravenous interventions and allow for easy blood collection.
- If owners are capable of care and monitoring at home, outpatient treatment of puppies that are not critically ill can be successful.

INTRODUCTION

Canine parvovirus (CPV) is one of the most common infectious diseases in dogs throughout the world, and proper vaccination is the most important strategy for prevention. It is highly contagious and spreads through the fecal–oral route. Treatment and prognosis are highly variable based on the severity of illness, and without treatment, the risk for death is high. With treatment, the chance for survival may be greater than 90%.

Parvoviruses are small, nonenveloped, single-stranded DNA viruses.[1] CPV belongs to the Parvoviridae family and genus *Protoparvovirus*. It infects mitotically active, rapidly dividing cells, including the gastrointestinal (GI) tract cells, bone marrow, lymphoid tissue, and cardiac myocytes. It has been suggested that CPV-1 may have been derived from feline panleukopenia virus.[1] CPV-1 mutated to the CPV-2 strain in the mid-1970s, leading to the first worldwide outbreak in the late 1970s.[2] CPV-2a, CPV-2b, and CPV-2c are the 3 variants currently described; they are clinically indistinguishable from one another.

ETIOLOGY AND PATHOPHYSIOLOGY

CPV is spread via the fecal–oral route; fomites, such as bedding, bowls, and toys, play an important role in transmission of the disease. It can survive for months in cool and

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Abbreviations	
CPV	canine parvovirus
ELISA	enzyme-linked immunosorbent assay
FFP	fresh frozen plasma
G-CSF	granulocyte colony-stimulating factor
GI	gastrointestinal
IV	intravenous
KCl	potassium chloride
MCV/L	mean corpuscular volume/lymphocyte count
NG	nasogastric
PCR	polymerase chain reaction
PDW/L	platelet distribution width/lymphocyte count
RDW/L	red cell distribution width/lymphocyte count
SQ	subcutaneous

moist environments.[1] Clinical signs may develop after the 4 to 7 day incubation period; however, the virus may be shed in the feces as early as 3 days after exposure.[1] Although viral shedding declines after approximately 1 week, it may continue for up to 4 weeks after infection.[1,3]

Viral replication occurs in the oropharyngeal lymphoid tissue, lymph nodes, and thymus, and viremia is seen 1 to 5 days after infection.[4] The virus is disseminated to rapidly dividing cells, such as the small intestinal crypt cells, bone marrow, thymus, lymph nodes, and cardiac myocytes.[3] The virus causes necrosis and villous collapse in the small intestinal crypt cells, resulting in impaired intestinal function, malabsorption, vomiting, and diarrhea. The compromised blood–gut barrier may allow bacterial translocation into systemic circulation. Destruction of the thymus and bone marrow precursor cells can lead to leukopenia. The combination of bacteremia and leukopenia may lead to sepsis or systemic inflammatory response syndrome. Puppies can develop myocarditis if infection occurs in utero or up to 2 weeks after birth. These puppies most commonly present at 3 to 8 weeks of age with congestive heart failure or after sudden death at home; they generally do not have GI signs.

Unvaccinated puppies between 6 weeks and 6 months of age are at the highest risk for the development of clinical signs associated with CPV infection. Dogs of any age can become infected, especially if they are unvaccinated or incompletely vaccinated; some of these dogs will not show clinical signs, even though they are shedding CPV in their feces. Specific breeds, such as Rottweilers, American pit bull terriers, Doberman pinschers, English Springer Spaniels, and German shepherds, may be at an increased risk for developing CPV.[5,6] Infection is more common during the warmer months, but depending on location, there may be a high risk throughout the year. This may be because puppies are more likely to be outside and interacting with other dogs during these times.

PREVENTION

The most effective way to prevent CPV is complete and timely vaccination. Maternal antibodies are present in puppies born from and those that nurse from vaccinated bitches. These maternal antibodies start to decrease at 8 to 12 weeks of age; therefore, CPV vaccinations are recommended to start at 6 weeks of age (**Table 1**). American Animal Hospital Association (AAHA) guidelines consider CPV a core vaccine. Dogs less than 16 weeks of age should receive at least 3 doses of a combination vaccine between 6 and 16 weeks of age, with 2 to 4 weeks between vaccinations.[7] Dogs aged older than 16 weeks should receive 2 doses of a combination vaccine, with 2 to

Table 1
Canine parvovirus vaccination schedule, starting at 6 wk of age

Age	Number of Doses	Frequency
<16 wk old	3 doses between 6 and 16 wk old	2–4 wk between vaccinations
>16 wk old	2 doses	2–4 wk between vaccinations
All dogs	1 dose administered 1 y after initial series	Boosters every 3 y

4 weeks between vaccinations.[7] One dose of a combination vaccine should be administered within 1 year after the initial vaccination series, and boosters should be administered every 3 years.[7] Following natural CPV infection, the puppy will likely have lifelong immunity to reinfection. However, CPV vaccination is generally part of a 3 way vaccine that includes immunization for canine distemper and adenovirus. Dogs will continue to require vaccination for the other components of the vaccine, and consequently, may continue to receive CPV vaccination. Despite the efficacy of CPV vaccines for preventing infection, any dog should be tested if it has clinical signs consistent with CPV.

If puppies are not completely vaccinated, contact with other dogs should be avoided. Fully vaccinated dogs without clinical signs can still shed CPV in their feces, so it is advised to avoid locations where CPV is likely to be present, for example, in parks. If CPV is in the environment or house, proper decontamination is required. CPV can be inactivated with a 1 in 30 dilution of bleach to water with a wet contact time of at least 10 minutes on nonporous surfaces.[1] Bedding and porous surfaces that cannot be decontaminated should be disposed of; steam cleaning can also be considered. Items such as bowls and dishes can be washed in dishwashers at a temperature of at least 75°C.[1] Shedding of CPV may continue for up to 4 weeks after infection, so quarantine is recommended, even if the dog appears to be feeling well.

Strict isolation, frequent handwashing, and appropriate personal protective equipment are required to prevent spread of CPV in a veterinary hospital. If a dog with CPV is brought into the general hospital, immediate cleaning and disinfection is recommended to reduce the risk of infection in other patients. After discharge from the hospital, appropriate cleaning and disinfection of the space and all equipment is required, including tables, bedding, fluid pumps, thermometers, and stethoscopes. Prior to disinfection, organic debris (including blood and feces) should be removed, and the area should be rinsed off. Ensure the disinfectant you are using is effective against CPV and determine if it can be used on porous and/or nonporous surfaces. A 1 in 30 dilution of bleach to water with a 10 minute wet contact time can be used. Alternatively, commercially available disinfectants that include potassium peroxymonosulfate (ie, Trifectant, 10 minute wet contact time) or accelerated hydrogen peroxide (ie, Rescue, 5 minute contact time) can also be used.

DIAGNOSIS

CPV infection is most commonly diagnosed using a fecal parvovirus antigen enzyme-linked immunosorbent assay (ELISA) test, performed with a rectal swab. There are multiple commercial assays available with varying sensitivities and specificities. False-negative results may be due to decreased viral shedding, especially early in disease. Recent vaccination with a modified-live vaccine can result in a false-positive antigen ELISA and polymerase chain reaction (PCR) test, especially 1 to 2 weeks postvaccination. However, any puppy with clinical signs or other diagnostic findings

consistent with CPV should be treated as having CPV, regardless of recent vaccination or negative ELISA testing. Although they are not routinely necessary, other diagnostic tests can be utilized to confirm a CPV infection. When the ELISA is negative and there is high suspicion of CPV infection, samples for PCR can be submitted.

CLINICAL SIGNS AND PHYSICAL EXAMINATION

Clinical signs develop 3 to 7 days after exposure. Severity of clinical signs can vary significantly; however, most puppies will present with some combination of lethargy, inappetence, vomiting, and diarrhea, which may be hemorrhagic. Myocarditis is uncommon and may present as peracute death or congestive heart failure in 3 to 8 week old puppies.

Physical examination will vary depending on the severity of clinics signs. Puppies may present in hypovolemic shock (intravascular fluid deficit), which can be diagnosed by evaluating perfusion parameters that include mentation, mucus membrane color, capillary refill time, heart rate, pulse quality, and extremity temperature. Abnormalities consistent with shock include obtundation, pale mucus membranes, prolonged capillary refill time, tachycardia (or bradycardia in decompensated shock), weak femoral pulses, and cold extremities. Puppies are commonly dehydrated (interstitial fluid deficit) with tacky mucus membranes and a prolonged skin tent. Rectal temperature may vary significantly with some puppies being normothermic, some febrile, and some hypothermic. Puppies may be painful on abdominal palpation, and fluid-filled intestines or a mass lesion due to intussusception may be noted. Diarrhea, which can be hemorrhagic, may be found on rectal examination. Severe obtundation or seizures may be seen in hypoglycemic puppies. Puppies with congestive heart failure secondary to myocarditis will be tachypneic with crackles on auscultation.

DIAGNOSTICS

Initial diagnostics in puppies with CPV may include a fecal parvovirus antigen ELISA, complete blood count (CBC), and chemistry panel. A bedside packed cell volume, total solids, glucose concentration, and venous blood gas can provide timely results in patients with more severe clinical signs or signs of shock. Blood pressure may be performed in patients with signs of shock. Coinfection with parasites is common, so fecal examination may be performed. When appropriate, the clinician should review pediatric reference intervals so that diagnostic results are not misinterpreted.

Leukopenia due to neutropenia and lymphopenia are common abnormalities identified on CBC, which may be seen on presentation or may develop during hospitalization. Leukopenia may be due to replication of the virus in rapidly dividing white blood cells or from sepsis. Some puppies will present with a neutrophilic leukocytosis. Puppies have a lower PCV than adult dogs, so they may appear anemic based on adult reference intervals. Puppies may also be anemic due to GI blood loss.

Hypoalbuminemia and hypoproteinemia are common in puppies with CPV due to loss through the GI tract; puppies also have a lower albumin concentration than adults. Hypoglycemia may be due to sepsis or due to decreased caloric intake with limited glycogen stores that are easily depleted. Hyponatremia, hypochloremia, and hypokalemia may be due to hypovolemia, vomiting, and decreased caloric intake. Prerenal azotemia may be seen in severely dehydrated or hypovolemic puppies. Venous blood gas may identify a metabolic acidosis due to hyperlactatemia (due to hypoperfusion), azotemia, or bicarbonate loss in diarrhea. A metabolic alkalosis may be present due to vomiting.

A coagulation panel and abdominal radiographs or ultrasound may be considered. Coagulation abnormalities have been reported in CPV puppies[8,9]; however, the clinical

relevance of these changes are unclear because they do not generally require specific treatment. Abdominal radiographs and ultrasound are not routinely recommended because they commonly show signs of nonspecific enteritis and ileus, and performing these diagnostics may increase the risk of contamination in the hospital. Abdominal imaging should be performed in patients when the clinician suspects a small intestinal obstruction due to intussusception or foreign body, in patients with severe abdominal pain that is not responsive to analgesics, and in patients with refractory vomiting.

TREATMENT

Dogs with CPV should be examined and treated in isolation, following strict biosecurity protocols. The severity of illness will dictate whether in-patient or outpatient treatment is recommended; the owner's ability to pay for treatment will also impact this decision. Treatment is largely supportive until the patient's clinical signs resolve. Critically ill patients may require prolonged hospitalization, which may come with significant costs to the client. Complications of CPV may include aspiration pneumonia, sepsis, and intestinal intussusception, so monitoring for these complications is recommended.

Vascular Access and Fluids

In patients with severe hypovolemia, placing an intravenous (IV) catheter may be difficult without a vascular cutdown. In cases when IV access cannot be obtained, an intraosseous catheter should be considered. Placement of a multilumen central venous catheter is highly recommended because these puppies often require many interventions and frequent blood sampling. A jugular catheter is preferred; however, a forelimb catheter is preferred over a hind limb catheter due to the risk for fecal contamination. Hypovolemic shock should be treated with administration of a 10 to 20 mL/kg bolus of an isotonic crystalloid (PlasmaLyte-A, lactated Ringer's solution, Normosol-R) without additives over 10 to 15 minutes (**Table 2**). A bolus can be repeated until normalization of perfusion parameters is achieved. Point-of-care ultrasound including cardiac and caudal vena cava views can also be used to monitor intravascular volume and response to treatment with fluid boluses. A 5 mL/kg bolus of fresh frozen plasma (FFP) may also be considered if the patient is not responding to repeated boluses of an isotonic crystalloid. Once the patient is euvolemic, the patient can be started on a rate of IV fluids that accounts for maintenance requirements, dehydration, and ongoing losses. Maintenance fluid requirements are higher for puppies (90–100 mL/kg/d), and these puppies often require high rates of fluids due to significant dehydration and ongoing GI losses. FFP can also be used during the maintenance phase, and it has the added benefit of containing proteins that provide oncotic support. While the administration of FFP is unlikely to increase the albumin concentration significantly, it may help maintain current concentrations. It will also limit the volume of crystalloids required, which may be beneficial for many reasons. A commonly reported maintenance dose of FFP is approximately 20 mL/kg/d, but this may vary widely depending on patient needs.

Dextrose and Potassium Supplementation

Puppies with CPV may become hypoglycemic; this can be treated with administration of 50% dextrose (0.5–1 mL/kg, diluted 1:4, administered over 1–2 minutes). After a slow bolus, 50% dextrose should be added to the bag of isotonic crystalloids. Adding 50 mL of 50% dextrose to a 1 L bag of fluids creates a 2.5% dextrose solution; adding 100 mL of 50% dextrose to a 1 L bag of fluids creates a 5% dextrose solution.

Table 2
Treatment considerations for canine parvovirus

IV fluids-hypovolemia	LRS, PlasmaLyte-A, Normosol-R	10–20 mL/kg over 10–15 min
IV fluids-hypovolemia	Fresh frozen plasma	5 mL/kg over 10–15 min
IV fluids-dehydration	LRS, PlasmaLyte-A, Normosol-R	Include maintenance, dehydration, and ongoing losses
IV fluids-maintenance	Fresh frozen plasma	20 mL/kg/d
Glucose	50% dextrose	0.5–1 mL/kg (diluted 1:4); 2.5%–5% dextrose in fluid bag
Potassium	KCl	0.2 mEq/kg/h; adjusted based on K^+ concentration
Antibiotics	Ampicillin-sulbactam	22 mg/kg IV q8 h
Antibiotics	Cefoxitin	22 mg/kg IV q6 h
Antinausea	Maropitant	1 mg/kg IV q24 h
Antinausea	Ondansetron	0.5–1 mg/kg IV q8 h
Prokinetic	Metoclopramide	2 mg/kg/d CRI
Enteral nutrition	Calorically dense food	Start at one-fourth to one-third of RER and increase q12–24 h
Analgesic	Buprenorphine	0.01–0.02 IV q6-8 h
Antioxidant	N-acetylcysteine	70 mg/kg IV q24 h × 5 d
Oral fluids	Oral recuperation fluids	Offered free choice
Microbiota support	Fecal transplant	10 g feces diluted with saline administered into the rectum
Antibody treatment	CPV monoclonal antibody	0.2 mL/kg IV slowly
Deworming	Deworming	Fenbendazole 50 mg/kg PO q24 h × 5 d

When puppies are hypokalemic, potassium chloride (KCl) can be added to the bag of isotonic crystalloids. KCl can be supplemented at 0.05 to 0.5 mEq/kg/h. Potassium supplementation at 0.05 mEq/kg/h is considered maintenance, and 0.5 mEq/kg/h is the maximum safe rate to administer potassium. This rate should only be used in patients that are severely hypokalemic or in patients that are suffering consequences of hypokalemia. If 0.5 mEq/kg/h is used, it should be administered for a maximum of 2 hours before the potassium concentration is re-evaluated. Most puppies with mild-to-moderate hypokalemia can start KCl supplementation at 0.2 mEq/kg/h, and the potassium can be closely monitored until it normalizes.

Antibiotics

Antimicrobial stewardship is critical in both human and veterinary medicine. While antibiotics are routinely prescribed in veterinary medicine, there are consequences to their use, including disruption of the intestinal microbiota and the development of resistant bacteria. Diarrhea or hemorrhagic gastroenteritis alone is not an indication for antibiotic prescription as it does not change the clinical course or outcome for

these dogs.[10–13] However, CPV puppies may not have a functional immune system due to leukopenia and neutropenia, and this may increase their risk for serious illness due to bacterial translocation. Therefore, when puppies are leukopenic or neutropenic, antibiotic administration is recommended. A penicillin/beta-lactamase inhibitor (ie, ampicillin-sulbactam 22 mg/kg IV q8h) or a cephalosporin (ie, cefoxitin, 22 mg/kg IV q6–8h) are commonly recommended. When CPV puppies are not leukopenic or neutropenic, antibiotics may not be necessary. The leukocyte count should be monitored because puppies may develop leukopenia during hospitalization. Antibiotics should also be prescribed in cases where the leukocyte count cannot be monitored, for example, if outpatient treatment is pursued, as these puppies may become septic and require antibiotics.

Sepsis should be suspected in puppies that remain hypotensive despite volume resuscitation. Other common signs of sepsis are hypoglycemia and leukopenia; these are also common signs in nonseptic CPV dogs, so they may be less useful in identifying septic dogs. In cases of sepsis, either a penicillin/beta-lactamase inhibitor and a fluoroquinolone or a penicillin/beta-lactamase inhibitor and an aminoglycoside should be considered. Fluoroquinolones may cause cartilage damage in young, large breed dogs, so the risk versus benefit should be weighed in these cases. Aminoglycosides may be nephrotoxic; therefore, the dog should be euvolemic and well-hydrated before administration, and the risk for acute kidney injury must be weighed.

Gastrointestinal Support

Vomiting, diarrhea, and inappetence are the most common clinical signs associated with CPV, so a multimodal approach to the treatment of GI signs is imperative. Antinausea medications, such as maropitant (1 mg/kg IV q24 h) and ondansetron (0.5–1 mg/kg IV q8h), should be administered. CPV commonly causes ileus, so the use of a prokinetic, such as metoclopramide (2 mg/kg/d; delivered as a constant rate infusion) or cisapride (0.1–0.5 mg/kg PO q8h), should be considered. Proton pump inhibitors (ie, pantoprazole or omeprazole) are not recommended for routine gastroenteritis, but they should be provided if there is concern for esophagitis. Gastric ulceration is uncommon in CPV puppies but is another indication for the prescription of proton pump inhibitors.

Enteral Nutrition

Enteral nutrition is essential in the treatment of CPV puppies because it is the only way to nourish the damaged enterocytes. Enteral nutrition helps maintain the GI barrier function by improving the integrity of the tight junctions and reducing catabolism. In one study, CPV puppies that received early enteral nutrition had a more rapid normalization of appetite, improved clinical scores, and improved weight gain compared to CPV puppies that were not fed until vomiting ceased for 12 hours.[14] Most puppies are not eating voluntarily, so a nasogastric (NG) tube should be considered. This allows for the controlled delivery of nutrition and removal of gastric residual volumes, which can contribute to discomfort and nausea and may increase the risk for regurgitation. A calorically dense liquid diet is preferred as it is less likely to clog the NG tube and limits the volume that needs to be administered. Liquid nutrition can be delivered through the NG tube as a constant rate infusion (CRI) or intermittent boluses. If an NG tube cannot be placed, syringe feeding is an option, but it may increase the risk for aspiration pneumonia.

Puppies have higher resting energy requirements, so it is commonly recommended to increase the RER to 1.5 times the adult calculation (daily resting energy requirements [RER] = $BW^{0.75} \times 70$). In most cases, the clinician should first prescribe one-

fourth to one-third RER and monitor closely for food intolerance, which includes intractable vomiting or regurgitation. Nutrition can continue to be delivered if the patient is intermittently vomiting or regurgitating, as long as the NG tube does not dislodge. The percentage of RER can be increased every 12 to 24 hours if it is well-tolerated.

Analgesics

CPV puppies should be prescribed analgesics for pain control. A partial mu-agonist, such as buprenorphine, is often sufficient. Full mu-agonists, such as methadone, fentanyl, or hydromorphone, are also options but may contribute to ileus. If a full mu-agonist is not sufficient to control pain, the patient should be evaluated for other causes of abdominal pain. Abdominal radiographs or an abdominal ultrasound can be considered to evaluate for a small intestinal obstruction or intussusception. If additional analgesics are necessary, a lidocaine (10–40 mcg/kg/min CRI) and/or ketamine (2–10 mcg/kg/min CRI) can be administered. Nonsteroidal anti-inflammatory drugs should not be given to CPV puppies in the acute timeframe due to GI signs and risk for acute kidney injury associated with hypovolemia and dehydration.

Other Treatment Considerations

N-acetylcysteine is an antioxidant that can be prescribed to treat oxidative stress. In one study, CPV puppies that received standard supportive care and N-acetylcysteine (70 mg/kg IV q24 h for 5 days) had significantly reduced markers of oxidative stress and improved leukocyte counts compared to CPV puppies that did not receive N-acetylcysteine.[15]

The use of hyperimmune fresh frozen plasma, made from dogs hyperimmunized against CPV and lipopolysaccharide (LPS), has been evaluated in dogs with CPV. Puppies that received standard supportive care and 10 mL/kg of hyperimmune FFP had significantly lower lactate and shock index heart rate/systolic arterial pressure (HR/SAP) at 24 hours compared to dogs that only received standard supportive care in one study. However, there was no difference in other traditional CPV endpoints, including duration of hospitalization, time to voluntary food intake, or survival to discharge.[16] It is unclear if the improved cardiovascular parameters at 24 hours was due to the patient receiving plasma or if it was due to the hyperimmune component of the plasma.

While there are multiple brands available, oral recuperation fluids commonly include prebiotics, omega fatty acids, amino acids, electrolytes, and vitamins. They are intended to promote enterocyte health, maintain hydration, and stimulate appetite. In one study, CPV puppies that were given an oral recuperation fluid had a more rapid return to voluntary food intake and a greater caloric intake at 24 hours compared to puppies who were not given the oral recuperation fluid.[17] Based on this study, a clinician may consider offering oral recuperation fluids in place of water.

The importance of the GI tract's microbiota cannot be underestimated, and many GI diseases are associated with its loss. Fecal transplants have been used in the treatment of a variety of GI diseases in an effort to restore the microbiota. In one study, CPV puppies that received a fecal transplant had a faster resolution of diarrhea and a shorter duration of hospitalization in those that survived.[18] The process for fecal transplantation is relatively easy; it involves the collection of feces (10 g) from a healthy, screened donor dog, diluting it with saline, and using a long catheter (ie, red rubber catheter) to deposit the sample in the proximal rectum. The pelvis can be raised for a few minutes to help retain the transplant.[18]

A new CPV monoclonal antibody treatment was recently approved by the Food and Drug Administration for the treatment of CPV. The treatment works by binding CPV in the bloodstream, preventing it from interacting with receptors on the target organ cells, such as the GI tract and bone marrow. It should be administered as early as possible in the course of disease because that is likely when viremia is highest. In an induced model of CPV in purpose-bred beagles, the administration of the antibody treatment was associated with a more rapid time to resolution of vomiting, lethargy, fever, lymphopenia, and inappetence.[19] In addition, there was a significant mortality benefit where all puppies that received the antibody treatment survived.[19] In this study, dogs in the control group did not receive any treatment, and dogs in the treatment group only received the antibody treatment, so it was not representative of how the antibody treatment is intended to be used clinically.

Human recombinant granulocyte colony-stimulating factor (G-CSF) has not been found to be beneficial in neutropenic CPV dogs.[20,21] Canine recombinant G-CSF has been evaluated with mixed results. In one study of neutropenic CPV puppies, neutrophil counts improved, but survival was decreased in puppies that received canine recombinant G-CSF.[22] In a second study, the administration of canine recombinant G-CSF was associated with improved white blood cell counts.[23] Recombinant feline interferon-omega has been evaluated in CPV puppies, and its use was associated with a more rapid improvement of clinical signs and reduced mortality.[24]

Parasitic infections are common in CPV dogs, so a deworming protocol, which may include fenbendazole (50 mg/kg PO q24 h for 5 days), should be initiated after vomiting has resolved. Specific parasites identified on fecal examination may require other treatments.

Outpatient Treatment

If dogs are not critically ill and the owners can provide appropriate monitoring and care at home, outpatient treatment can be considered (**Box 1**).[25] If the puppy is not improving, if it continues to have large volumes of diarrhea and/or vomiting, or if it is progressively lethargic, it should be re-evaluated by a veterinarian.

MONITORING AND CARE

Nursing care of CPV puppies is often extensive, with frequent needs for cleaning and feeding. It is important to ensure puppies are clean and dry to avoid fecal and urine scald. External heat support may be required, and frequent monitoring of temperature is recommended.

Vital signs and pain assessment should be performed at least every 6 hours. Body weight, arterial blood pressure, packed cell volume and total solids, blood glucose, and venous blood gas (which measures electrolytes and lactate) should be assessed every 12 hours. More frequent assessment may be needed in cases of significant electrolyte disturbances, persistent hyperlactatemia, or hypoglycemia. Leukocyte count should be assessed every 24 hours; this may be performed with a blood smear or CBC. In critically ill dogs, all monitoring may need to be performed more frequently. Frequent blood sampling can contribute to anemia in hospitalized dogs, so obtaining the minimum amount of blood for each test is recommended.

PROGNOSIS

The prognosis for CPV dogs will vary highly based on severity of illness and the ability to provide necessary supportive care. With appropriate treatment, survival may be over 90%. There are many factors that may be associated with mortality. CBC

Box 1
Outpatient treatment recommendations

Initial treatments provided in hospital prior to discharge.
- Hypovolemia → IV catheter and administer fluid boluses to achieve normovolemia
- Hypoglycemia → administer 50% dextrose IV to achieve euglycemia; 0.5 to 1 mL/kg diluted 1:4%; 2.5% to 5% dextrose in fluid bag
- Hypokalemia → supplement KCl in IV fluids
- Cefovecin: 8 mg/kg subcutaneous (SQ) once[25]
- Maropitant: 1 mg/kg IV or SQ[25]
- Buprenorphine: 0.01 to 0.02 mg/kg IV or SQ[25]
- SQ fluids; balanced, isotonic crystalloid: 30 mL/kg, demonstrated to owners[25]
- CPV monoclonal antibody: 0.2 mL/kg IV slowly may be considered

At-home treatment plan
- Subcutaneous fluids: 30 mL/kg q 6 hours[25]
  - Additional SQ fluids can be administered in the first 24 hours to treat dehydration
- Maropitant: 2 mg/kg PO q 24 hours; may consider SQ administration if oral medications are not tolerated[25]
- Buprenorphine: 0.01 to 0.02 mg/kg on the mucus membranes q 8 hours; consider SQ administration if oral medications are not tolerated[25]
- Potassium gluconate: 0.5 to 1 mEq/kg PO q12 hours if needed for treatment of hypokalemia[25]
- Nutrition: offer a calorically dense food every 6 hours; if puppy is not eating, syringe feeding should be attempted
  - If puppy is not swallowing or is gagging, stop syringe feeding
- High fructose corn syrup: 1 to 5 mL on the gums for signs of hypoglycemia
  - Immediate veterinary care should be sought
- Nursing care: keep the puppy clean and dry

parameters that have been identified as negative prognostic indicators include leukopenia and lymphopenia,[26,27] failure for leukopenia or lymphopenia to resolve within 24 to 72 hours,[28,29] higher neutrophil-to-lymphocyte ratio,[27] low hematocrit (HCT) at admission,[30] number of band neutrophils at 24 and 48 hours,[28] monocytopenia,[28] and eosinopenia.[28]

Other negative prognostic indicators that have been reported are low glucose concentration at admission,[30] chloride concentration less than 96 mmol/L,[31] increased magnesium concentration at admission,[30] low body weight,[26,30] presence of systemic inflammatory response syndrome,[32] increasing sequential organ failure assessment score over the first 4 days of hospitalization,[33] lower cobalamin concentrations,[34] higher c-reactive protein (CRP),[31] and elevated cortisol at 72 hours.[29] The positive predictive value of survival using multiple factors were reported in one study. The positive predictive value for survival was 89.5% for a total leukocyte count using a cutoff of greater than $3.2 \times 10^3/\mu L$, 88.9% for a neutrophil count using a cutoff of greater than $1.65 \times 10^3/\mu L$, and 89.5% for a cortisol concentration using a cutoff of less than or equal to 16.9 ng/mL.[29] There is also a model published that is used to predict survival based on antithrombin, aspartate aminotransferase, lipase, monocyte count, and lymphocyte count.[35] A publication by Sanaei[36] reported the utilization of machine learning models to predict clinical outcome and recovery time in CPV dogs. Finally, another study reported that red cell distribution width/lymphocyte count (RDW/L), mean corpuscular volume/lymphocyte count (MCV/L), mean platelet volume/lymphocyte count, platelet/lymphocyte count, plateletcrit/lymphocyte count, and platelet distribution width/lymphocyte count (PDW/L) ratios were significantly higher in septic dogs with CPV compared to healthy dogs.[37] In the same study, CRP and CRP/albumin were also higher in septic dogs compared to healthy dogs.[37] Additionally, the

sensitivity and specificity for predicting mortality were 95% and 80% for MCV/L, 100% and 80% for RDW/L, 100% and 80% for MPV/L, and 100% and 80% for PDW/L.[37]

CLINICS CARE POINTS

- Strict isolation, frequent handwashing, and appropriate personal protective equipment are required to prevent the spread of CPV in the hospital.
- IV fluids and enteral nutrition are critical in the treatment of patients with CPV. Enteral nutrition is most easily provided with the placement of an nasogastric tube.
- Nursing care requirements for CPV puppies is extensive, with frequent needs for cleaning and feeding.
- If puppies are not critically ill and owners can provide appropriate monitoring care at home, outpatient treatment can be considered. Intitial treatments to correct hypovolemia, hypoglycemia, and hypokalemia should be performed in hospital, prior to discharge.

DISCLOSURE

The author has served as a key opinion leader for Elanco, who produces Trutect, a CPV monoclonal antibody.

REFERENCES

1. Parrish CR, Sykes JE. Canine parvovirus infections and other viral enteritides. In: Sykes JE, editor. Greene's infectious diseases of the dog and cat. 5th edition. St Louis (MO): Elsevier; 2023. p. 341–51.
2. Hoelzer K, Parrish CR. The emergence of parvoviruses of carnivores. Vet Res 2010;41:39.
3. Goddard A, Leiswitz AL. Canine parvovirus. Vet Clin Small Anim 2010;40:1041–53.
4. Meunier PC, Cooper BJ, Appel MJG, et al. Pathogenesis of canine parvovirus enteritis: sequential virus distribution and passive immunization studies. Vet Pathol 1985;22(6):617–24.
5. Glickman LT, Domanski LM, Patronek GJ, et al. Breed-related risk factors for canine parvovirus enteritis. J Am Vet Med Assoc 1985;187(6):589–94.
6. Houston DM, Ribble CS, Head LL. Risk factors associated with parvovirus enteritis in dogs: 283 cases (1982-1991). J Am Vet Med Assoc 1996;208(4):542–6.
7. Ellis J, Marziani E, Aziz C, et al. AAHA canine vaccination guidelines. J Am Anim Hosp Assoc 2022;58(5):213–30.
8. Otto CM, Rieser TM, Brooks MB, et al. Evidence of hypercoagulability in dogs with parvoviral enteritis. J Am Vet Med Assoc 2000;217(10):1500–4.
9. Corda F, Ballocco I, Corda A, et al. Coagulation abnormalities in dogs with parvoviral enteritis. Vet Sci 2023;10(1):41.
10. Unterer S, Strohmeyer K, Krus BD, et al. Treatment of aseptic dogs with hemorrhagic gastroenteritis with amoxicillin/claculanic acid: a prospective blinded study. J Vet Intern Med 2011;25(5):973–9.
11. Reisinger A, Stubing H, Suchodolski J, et al. Comparing treatment effects on dogs with acute hemorrhagic diarrhea syndrome: fecal microbiota transplantation, symptomatic therapy, or antibiotic treatment. J Am Vet Med Assoc 2024;262-(12):1657–65.

12. Shmalberg J, Montalbano C, Morelli G, et al. A randominzed double blinded controlled clinical trial of probiotic or metronidazole for acute canine diarrhea. Front Vet Sci 2019;6:163.
13. Stubing H, Suchodolski JS, Reisinger A, et al. The effect of metronidazole versus symbiotic on clinical course and core intestinal microbiota in dogs with acute diarrhea. Vet Sci 2024;11(5):197.
14. Mohr AJ, Leisewitz AL, Jacobson LS, et al. Effect of early enteral nutrition on intestinal permeability, intestinal protein loss, and outcome in dogs with severe parvoviral enteritis. J Vet Intern Med 2003;17(6):791–8.
15. Gaykwad C, Garkhal J, Chethan GE, et al. Amelioration of oxidative stress using N-acetylcysteine in canine parvoviral enteritis. J Vet Pharmacol Ther 2018;41(1):68–75.
16. Acciacca RA, Sullivan LA, Webb TL, et al. Clinical evaluation of hyperimmune plasma for treatment of dogs with naturally occurring parvoviral enteritis. J Vet Emerg Crit Care 2020;30(5):525–33.
17. Tenne R, Sullivan LA, Contreras ET, et al. Top Companion Anim Med 2016;31(2):68–72.
18. Pereira GQ, Gomes LA, Santos IS, et al. Fecal microbiota transplantation in puppies with canine parvovirus infection. J Vet Intern Med 2018;32(2):707–11.
19. Larson L, Miller L, Margiasso M, et al. Early administration of canine parvovirus monoclonal antibody prevented mortality after experimental challenge. J Am Vet Med Assoc 2024;262(4):506–12.
20. Rewerts JM, McCaw DL, Cohn LA, et al. Recombinant human granulocyte colony-stimulating factor for treatment of puppies with neutropenia secondary to canine parvovirus infection. J Am Vet Med Assoc 1998;213(7):991–2.
21. Mischke R, Barth T, Wohlsein P, et al. Effect of recombinant human granulocyte colony-stimulating factor on leukocyte count and survival rate of dogs with parvoviral enteritis. Res Vet Sci 2001;70(3):221–5.
22. Duffy A, Dow S, Ogilvie G, et al. Hematologic improvement in dogs with parvovirus infection treated with recombinant canine granulocyte colony-stimulating factor. J Vet Pharmacol Ther 2010;33:352–6.
23. Armenise A, Trerotoli P, Cirone F, et al. Use of recombinant canine granulocyte colony-stimulating factor to increase leukocyte count in dogs naturally infected by canine parvovirus. Vet Microbiol 2019;231:177–82.
24. de Mari K, Maynard L, Eun HM, et al. Treatment of canine parvoviral enteritis with interferon-omega in a placebo-controlled field trial. Vet Rec 2003;152(4):105–18.
25. Venn EC, Preisner K, Boscan PL, et al. Evaluation of an outpatient protocol in the treatment of canine parvoviral enteritis. J Vet Emerg Crit Care 2017;27(1):52–65.
26. Dossin O, Rupassara SI, Weng HY, et al. Effect of parvoviral enteritis on plasma citrulline concentration in dogs. J Vet Intern Med 2011. 2011;25(1):215–21.
27. Gonzalez-Dominguez A, Cristobal-Verdejo JI, Lopez-Espinar C, et al. Restrospective evaluation of hematological ratios in canine parvovirus: 401 cases. J Vet Intern Med 2024;38(1):161–6.
28. Goddard A, Leisewitz AL, Christopher MM, et al. Prognostic usefulness of blood leukocyte changes in canine parvoviral enteritis. J Vet Intern Med 2008;22(2):309–16.
29. Eregowda CG, Kumarde U, Singh M, et al. Assessment of certain biomarkers for predicting survival in response to treatment in dogs naturally infected with canine parvovirus. Microb Pathog 2020;149:104485.

30. Chalifoux NV, Parker SE, Cosford KL. Prognostic indicators at presentation for canine parvoviral enteritis: 322 cases (2001-2018). J Vet Emerg Crit Care 2021; 31(3):402–13.
31. Paul BR, De UK, Sarkar VK, et al. Prognostic potential of thrombocyte indices, acute phase proteins, electrolytes, and acid-base markers in canine parvovirus infected dogs with systemic inflammatory response syndrome. Top Companion Anim Med 2023;56-57:100803.
32. Kalli I, Leontides LS, Mylonakis ME, et al. Factors affecting the occurrence, duration of hospitalization and final outcome in canine parvovirus infection. Res Vet Sci 2010;89(2):174–8.
33. Kalogianni L, Polizopoulou ZS, Kazakos G, et al. The role of the sequential organ failure assessment score in evaluating the outcome in dogs with parvoviral enteritis. Res Vet Sci 2022;150:44–51.
34. Luckschander-Zeller N, Giani B, Doulidis PG, et al. Implications of hypocobalaminemia as a negative prognostic marker in juvenile dogs with parvovirus enteritis. Front Vet Sci 2024;11:1426664.
35. Franzo G, Corso B, Tucciarone CM, et al. Comparison and validation of different models and variable selection methods for predicting survival after canine parvovirus infection. Vet Rec 2020;187(9):e76.
36. Sanaei N, Zamani-Ahmadmahmudi M, Nassiri SM. Development of machine learning models to predict clinical outcome and recovery time in dogs with parvovirus enteritis. Front Vet Sci 2025;12:1555714.
37. Varlik T, Algan D, Ceron J, et al. The diagnostic and prognostic utility of complete blood count derived indices in dogs with sepsis caused by canine parvoviral infection. Vet Res Commun 2025;50(1):11.