# **The Parvo puppy**

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Canine parvovirus (CPV) is commonly seen both in shelters and in pet dogs and remains a significant worldwide canine pathogen.

CPV is a nonenveloped virus. Because a viral envelope is so fragile, viruses which do not

have to rely on this structure (nonenveloped viruses), such as CPV, are very hardy. This

means that good biosecurity is essential in the control of CPV, as fomite spread is so important.

CPV has a DNA genome, which means it is stable and mutates relatively rarely. However,

over the last 40 years, there have been a number of mutation events which have been important in changing the host range of this virus.

1. **Pathophysiology**

Canine parvovirus often infects unvaccinated puppies between 6 weeks and 6 months of age. Breed, genetic background, and the innate immune system all factor into individual response to infection. It is likely that both genetic and environmental factors impact risk of CPV infection for a given individual. Lack of vaccination poses the greatest threat to puppies exposed to CPV. An incubation period of 4-10 days follows oronasal exposure to CPV-contaminated feces. Virus replication occurs in lymphoid tissue of the oropharynx, thymus, and regional lymph nodes. Viremia occurs within 1–5 days of infection, facilitating CPV movement into the germinal epithelium of enterocytes. Small intestinal crypt cells necrose and cause villous collapse, leading to altered gastrointestinal tract function. The compromised blood-gut barrier encourages movement of gram-negative and anaerobic bacteria from the intestinal lumen into the bloodstream. Secondary bacteremia may result in life-threatening complications, including sepsis and coagulation disorders.

1. **Clinical presentation**

Non-specific signs of illness such as lethargy and anorexia may be noted in the peracute phase of infection. Following this brief period, symptoms of enteritis predominate. Classic symptoms of CPV include anorexia, vomiting, and diarrhea.

Severe vomiting and diarrhea lead to dehydration, hypovolemia, and cardiovascular collapse. Dogs often present with abnormal perfusion parameters including tachycardia, abnormal mucous membrane color/capillary refill time, weak or absent pulses, and altered mentation. Most CPV patients display moderate to severe abdominal pain. This visceral pain is most likely associated with diffuse enteritis, although intestinal intussusception should remain a differential diagnosis for severe pain. Mentation changes and generalized weakness may be due to hypoperfusion, hypoglycemia, hypokalemia, or any combination thereof. Although the gastrointestinal tract is primarily involved with CPV infection and transmission, other tissues may be affected. Myocardial disease has been reported with neonatal infection; myocarditis may precede intestinal signs and result in peracute death in such cases (Sime et al. 2015). Other organs affected by CPV may include the respiratory system, liver, and kidneys.

1. **Diagnostic test**

An enzyme-linked immunosorbent assay (ELISA) is available to detect viral antigen in feces or rectal swab for all CPV type-2 variants. In some cases, false positives or false negatives can occur. However, a positive test associated with clinical signs such as vomiting, diarrhea, lethargy and lack of appetite supports the diagnosis of parvovirus. False-positive CPV ELISA results following modified live virus vaccination were not demonstrated in dogs from a recent study. Other diagnostic tests, such as rectal swab PCR, are available, but take a longer time to get results.

Minimum diagnostics should include a packed cell volume and total protein, blood glucose, as well as a blood smear and electrolytes to screen for leukopenia, thrombocytopenia, hypokalemia, azotemia, and hypoglycemia. A fecal flotation is ideal, although empirical deworming is often pursued with treatment.

White blood counts for parvovirus are generally described as low to very low. This result is due to the destruction of the hematopoietic progenitors cells of the various leucocyte lineages in the bone marrow as well as to the sequestration of leucocytes in the necrotic intestinal cells of the crypts. However, leucopenia is found in only 50% of parvovirus dogs. Thus, leukopenia is neither a sensitive nor specific indicator of parvovirus infection in infected dogs. While most clinicians are interested in the neutropenia of infected dogs, it has recently been shown that an absolute value of less than 1000 lymphocytes / μl within 48 hours of admission is a negative prognostic indicator. Other (non-specific) biological changes that can be observed with parvovirus infection include: hypoproteinemia, hypoalbuminemia, anemia, extrahepatic cholestasis, pre-renal azotemia, coagulation abnormalities, including thrombocytopenia and prolonged clotting times, hypoglycemia and hypokalemia. Significantly increased levels of plasma proteins that mark the inflammatory phase (C-reactive protein and ceroplasmin) have been found in dogs with parvovirus, and may help predict mortality.

1. **Treatment and monitoring**
2. **Fluid therapy**

Fluid therapy is the cornerstone of the treatment.

Points that need to be assessed at admission, are, in this order:

* Is this puppy hypovolemic?
* Is this puppy dehydrated?
* Does this puppy show any water loss?
* What is the maintenance of this puppy?

Hypovolemia is the first abnormality to assess. Isotonic crystalloids are the fluids of choice, and should be given IV in bolus of 10 to 20 ml/kg over 10 min. Cardiovascular parameters should then be reassessed, and bolus administered until normalization of volemia. Colloïdal and hypertonic support can be considered, but evaluation of risks should be done before (coagulopathy, hypernatremia…) In case of persistent hypovolemia, vasoactive drug such as norepinephrine should be started.

Once perfusion parameters have normalized, a maintenance fluid plan should commence. Calculating a maintenance fluid rate includes the following components:

* Correction of dehydration: (MilliLiters to replace = % dehydration x 10 x body weightkg). This volume should be replaced over a period of 12–24 hours.
* Ongoing insensible losses (estimated and replaced every 4–6 hours as an isotonic fluid) due to vomiting and diarrhea.
* Ongoing sensible losses: in a puppy, maintenance fluid is 70 ml/kg/d for normal urinary, fecal, and respiratory losses.

Fluid therapy should also address metabolic and electrolytic abnormalities. Hypoglycemia and hypokalemia are the most frequent and life-threatening condition. If the blood glucose of a symptomatic CPV puppy is found to be ≤ 60 mg/dL, an IV dextrose bolus should be administered immediately. The recommended dose of 50% dextrose is 0.5–1 mL/kg (0.25–0.5 g/kg) diluted 1:2 with isotonic saline. Additional dextrose boluses can be administered if needed, and dextrose may be placed in the base fluids as a CRI during the maintenance fluid

stage. Potassium supplementation of fluids, guided by regular monitoring of serum potassium concentration, as part of the daily fluid plan is an important part of management of the CPV patient. Severe hypokalemia may need a continuous infusion of KCl. Magnesium is an important co-factor in potassium homeostasis, and supplementation may help restore normal serum potassium levels rapidly.

1. **Antiemetic**

Antiemetics may be necessary in case of severe vomiting. The two most commonly used drugs are metoclopramide (1-2 mg / kg / day IV CRI) and maropitant (1 mg / kg IV q24h). IV antacids can be added (pantoprazole, 1 mg / kg IV q24h).

1. **Antibiotic**

Intravenous bactericidal antibiotics are indicated in CPV puppies with leukopenia. It is recommended to use the narrowest antimicrobial spectrum and shortest treatment duration possible. Single-agent antibiotic options include penicillins with beta-lactamase inhibitor activity (e.g. ampicillin-sulbactam) and second-generation cephalosporins. In the authors’

hospitals, single-agent ampicillin-sulbactam is frequently used at a dose of 30 mg/kg IV q8 h.

The presence of gastrointestinal parasites can aggravate the morbidity of patients and compromise the response to treatment. The search for Giardia, coccidia, Isospora and other gastrointestinal parasites is strongly recommended as well as empirical deworming in the most severe cases.

1. **Enteral nutrition**

Enteral feeding should be started as soon as possible with nasal feeding tube, even if vomiting is still present. Benefits of naso-gastric tubes is the ability to intermittently suction gastric contents. Removal of stagnant gastric fluid appears to reduce nausea and quantification of these ongoing losses can assist in tailoring fluid therapy.

Resting energy requirement are calculated as: 70 x BW0.75 (kg). Small amount of food could be given the first day, and increased based on the patient tolerance.

1. **Analgesia**

Dogs with CPV demonstrate moderate-to-severe visceral pain.

Multimodal analgesia is used according to the dog's pain score. Continuous rate infusions (CRI) of opioids can be used for severe pain. They can consist of:

* Morphine: 0.1 to 0.3 mg / kg / h or fentanyl: 1 to 5 μg / kg / h (may cause ileus)
* Lidocaine: 25 to 50 μg / kg / min
* Ketamine: 10 μg / kg / min

Buprenorphine (0.02 mg / kg IV q6 at 8h) is interesting for moderate pain. Non-steroidal anti-inflammatory drugs should be avoided, and alpha-adrenergic agonists should be used with caution in these patients.

1. **Fecal microbiome transplantation**

Fecal microbiome transplantation (FMT) entails the transfer of feces from a healthy donor into the intestinal tract of a diseased recipient. It has been associated with faster resolution of diarrhea in parvovirus-infected puppies in a randomize controlled study. Description of the technique can be found in the paper from Pereira et al. (JVIM 2018, DOI: 10.1111/jvim.15072).

In the author institution, FMT is used for one year now, and as shown very promising results. This is easy to do, inexpensive, and with no adverse effect.

1. **Additional treatment**

Canine plasma products have been previously advocated in the treatment of CPV, citing provision of antibodies and coagulation factors, in addition to oncotic support. However, considering the risks and cost associated with plasma transfusion, routine use of plasma in

CPV puppies is not recommended. In cases of disseminated intravascular coagulation with overt hemorrhage, fresh frozen plasma may be administered at a dose of 10–20 mL/kg.

Nursing care are very important for parvo puppies. As they are immunocompromised, be sure that they are clean is mandatory. Patients with CPV should be isolated while in the hospital because of the highly contagious nature and hardiness of the virus.

1. **Outpatient protocol**

While hospitalization is ideal for treating most CPV patients, in-hospital therapy may not be feasible for some owners or in certain clinical settings. Outpatient protocols consisting of subcutaneous fluid therapy, antiemetics, and antimicrobials, along with oral dextrose, oral potassium, and syringe feeding have been described (JVECC 2017, doi: 10.1111/vec.12561, JAVMA 2017, doi: 10.2460/javma.251.9.1035 ).

1. **Prevention**

Vaccination is critical in the prevention of CPV infection. Canine parvovirus vaccine is considered a core vaccine by the American Animal Hospital Association. Vaccination with a modified live vaccine starting as early as 6 weeks of age and continuing every 2-4 weeks until at least 16 weeks of age is recommended. The last vaccine in the series should be given at 16 weeks of age to avoid maternal antibody interference. Vaccination with vaccines containing CPV-2b produces immunity against CPV-2a and CPV-2c. For further information on canine parvovirus vaccination, see AAHA’s 2017 Canine Vaccination Guidelines.