Canine Parvovirus Enteritis

**CLINICAL SIGNS**
- Vomiting
- Malaise
- Abdominal pain
- Anorexia
- Pyrexia
- +/- Diarrhoea
- +/- Hematochezia

**DIAGNOSTICS**
- Parvovirus test
- PCV/TSP
- Blood glucose
- Electrolytes + blood gas
- CBC
- Biochemistry
- +/- SNAP Giardia test, fecal float

**HISTORY**
- Seen commonly in dogs less than 6 months, generally between 6 weeks and 6 months of age
- Certain breeds have been found to have a predisposition for severe infection, including Rottweilers, Doberman Pinchers, American Pit Bull Terriers, Labrador Retrievers and German Shepherd dogs.
- A distinct seasonality has been reported with peak incidence of disease occurring in the summer months

**PATHOPHYSIOLOGY**
Parvovirus [CPV-1] was first discovered in the late 1960s and caused mild diarrhoea. About a decade later, a new species of Paroviridae emerged in the US, which caused severe enteric disease and had a high morbidity and mortality in the naïve canine population. This new species was named canine parvovirus type 2 [CPV-2]. The virus continued to mutate, producing subtypes CPV-2a, CPV-2b, and most recently CPV-2c. Today, CPV-2a and CPV-2b are the most common parvovirus species causing disease in canine populations globally. Conventional type 2 vaccines appear to be efficacious in dogs challenged experimentally with CPV-2c.

Parvovirus [genus: Paroviridae] are small, non-enveloped, single-stranded DNA viruses that typically replicate in the nucleus of dividing cells in late S phase or early G2 phase of the cell cycle. Viral replication results in cell death and loss due to failure of mitosis. The virus has a predilection for rapidly diving cells such as intestinal epithelium, bone marrow, lymphoid cells, and myocardium [rare] in young puppies <4 weeks. Effective immunization against the disease is essential in the prevention of infection in susceptible puppies. Maternal antibodies are transferred in colostrum and placenta and the amount of antibody transferred is variable due to litter size and titre present in the dam. In pups that received colostrum from dams with a low antibody titre, protection may be only present for 4-6 weeks after birth. In dams with high antibody titres, protection may last from 12-20 weeks of age in the pup and is the reason why at least 3 vaccinations are recommended (maternal antibodies can negate effects of vaccine. The half-life of the maternal antibody was reported to be 10 days.

CPV is spread directly via the faecal-oral route, or indirectly via oro-nasal exposure to fomites contaminated by faeces. The virus first replicates in the oropharyngeal lymphoid tissue, mesenteric lymph nodes, thymus, and then spreads haematogenously. By 3-4 days post-infection, the virus enters the intestinal crypt cells causing epithelial destruction and villous collapse, and subsequent shortened and atrophic villi. Antibodies are produced by day 5 of infection and peak at days 7-10. Virus has been isolated from lungs, spleen, liver, kidneys and...
Guidelines:

Class 1 (C1) – Definitely perform (good evidence)

Class 2 (C2) – Consider performing (some evidence)

Class 3 (C3) – Do not perform (unsound evidence and/or deleterious)

AES Protocols Excellence Program (Canine Parvovirus Enteritis)
• Loss of antithrombin through the GI tract, as well as consumption as a result of endotoxin-mediated activation of coagulation and hyperfibrinogenemia are thought to contribute to the hypercoagulable state seen in CPV enteritis patients

**Biochemistry + electrolytes**

• Hypoglycemia may be a result of immature hepatic function, sepsis, inadequate glycogen stores and/or decreased caloric uptake
• May see hyperbilirubinemia, elevated liver enzymes, hypokalemia and a pre-renal azotemia
• Hypoalbuminemia is also a negative acute phase protein and production is down regulated in states of systemic inflammation [SIRS]

**+/- Diagnostic imaging : Ultrasound useful for early detection of concurrent intussusception. It should be considered as part of the diagnostic investigation, but performed in all cases where acute deterioration occurs, there is a palpable abdominal abnormality, or failure to improve over 48 hours.**

• Abdominal ultrasound findings which are indicative of CPV enteritis include: altered wall layering of the duodenum and jejunum with hyperechoic mucosal speckling and undulation of the luminal mucosal surface, thinning of the duodenal and jejunal mucosa without significant decrease in overall wall thickness and generalized hypomotility to immotility with fluid-filled intestinal lumen.
  *Intussusception is an important differential for CPV enteritis or can be a potential sequela of the disease.*

**TREATMENT**

**INITITAL MANAGEMENT**

**FIRST 24H:**

**Fluid therapy**

• Buffered replacement crystalloid fluids (hartmans or P148) should be used for initial fluid resuscitation
• The use of plasma for volume expansion and treatment of hypoalbuminaemia is controversial but should be considered. The small size of most parvovirus patients means that FFP can significantly affect volume, and albumen level. Consider use if patient is hypoalbuminaemic and coagulopathic, or hypovolaemic and does not respond to crystalloid ressuscitation
• Most patients will require supplementation of fluids with Potassium. If hypoglycaemic add 50% dextrose to fluids to make up a 2.5%, or 5% solution.

**Antibiotic therapy: 4 Quadrant antibiotic therapy is indicated in septic patients**

• Patients with evidence of sepsis, pyrexia, haematochezia or severe neutropenia require antibiotic therapy
• Ampicillin 20mg/kg IV q8hr (C1) alternately Cephazolin 22mg/kg IV Q 8h (or other 1st generation cephalosporins) (C2)
• Enrofloxacin 5mg/kg SC q24hrs (consider effects on cartilage development for extended use) (C2)
• +/- Metronidazole if parasitic co-infection present 10mg/kg IV q12h (C2)

If sepsis persists, or recurs, blood cultures should be performed, and antibiotic therapy escalated consider in order of preference (depending on availability):

• Ticarcillin/clavulanate combination (Timentin)
• Amikacin 20mg/kg IV, SC or IM q24h (C2)

_Antiemetic therapy_

• Maropitant 1mg/kg SC q24 h (5 days only) (C1)
• +/- Onodansetron 0.1-0.176 mg/kg slow IV q6-12h when vomiting is unresponsive to other drugs (C2)
• +/- Metoclopramide 0.2-0.5mg/kg q6h IV then 1-2mg/kg/day added to crystalloid fluids – only when GI obstruction ruled out (C2)

_Gastric protectants_

• Esomeprazole 1.0mg/kg IV q12hrs (C1)

_Analgesia_

• Buprenorphine 0.01-0.03mg/kg IV q6-8hrs (C1)
• +/- Lignocaine CRI at 1-3mg/kg/hr (C2)
• +/- Methadone 0.1-0.3mg/kg SC q4-6hrs (care with profound sedation and ileus) (C2)

_Nutrition (If not eating on day 1, start enteral nutrition immediately)_

• Encourage feeding with small frequent meals of a low-fat, easily digestible diet (eg: a/d slurry) (C1)
• Placement of a nasooeosophageal tube for enteral feeding (C1)
• Start enteral nutrition ASAP (C1)
  o Lectade CRI at 0.5ml/kg/hr, then increase to 1ml/kg/hr to monitor response initially
  o If lectade is well tolerated, start Ensure CRI at 1/3 RER and gradually increase q6-12hrs by 1/3 RER

*Enteral nutrition helps to maintain intestinal integrity and decreases bacterial translocation

_Barrier nursing_

• CPV-infected patients, or suspect CPV infected patients should be held in isolation during their time in hospital
• Pens should be cleaned with viricidal agents such as 1 part bleach or F10 to 32 parts water

_Controversial treatments_

• The use of anti-endotoxin has been suggested, however published studies have had conflicting results in regards to survivability (not recommended at AES)
• Granulocyte stimulating factor (G-CSF) has not been shown to improve survivability in CPV cases but is very expensive (not recommended at AES)
• Administering CPV-immune plasma to create passive immunity has been studied, however there were no significant differences in leucocyte count, duration of hospitalization, cost of overall treatment and magnitude of viremia among the treatment and placebo groups. (FFP is not recommended for this purpose at AES- see indications above)

_Out-patient treatment protocol_

• A study published in JVECCS in 2017 showed a reasonable prognosis for parvovirus patients who could not afford inpatient treatment (see ref).
• The key aspects of the outpatient treatment protocol were:
• Administration of subcutaneous (SC) fluid (30 mL/kg q 6 h) a
• Administration of maropitant (1 mg/kg SC q 24 h) and cefovecin (8 mg/kg SC once).
• Daily dextrose and potassium supplementation was provided orally (outpatients) as indicated (daily measurements were collected).
• Rescue criteria were used for analgesia and nausea.
• All dogs were syringe fed a commercial canine convalescence diet (1 mL/kg PO q 6 h) until voluntary appetite returned.

*This protocol should only be considered in mild to moderately affected patients if euthanasia is the only option. It is not an alternative to inpatient treatment of this disease and significant costs will still be incurred by the pet owner, as well as requiring daily revisits to hospital until the pet has recovered.*

**COSTS AND HOSPITALISATION**

• Hospitalisation time to expect: 3-7 days
• Costs whilst hospitalized: $4000-$10000

**PROGNOSIS AND RISK FACTORS**

• Survival rates in dogs treated for canine parvovirus vary widely [64-92%]
• Factors that predispose to CPV infection in puppies include: lack of protective immunity, intestinal parasites, and overcrowded, unsanitary and stressful environmental conditions
• An animal that has recovered from CPV infection should be kept away from other dogs initially for at least 14 days as the animal continues to shed and owners should be mindful of carefully disposing of faeces.
• Virus shedding can continue for up to 39 days post-infection

**REFERENCES**

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