Canine Parvovirus (CPV) is the greatest viral threat to dogs, in Australia. Every year thousands of dogs and puppies are infected with approximately half of these cases dying, either directly from the virus, or indirectly due to euthanasia due to cost of treatment, or for humane reasons.

During CPV outbreaks, veterinarians are often confronted with cases of disease that may not be considered a typical presentation. CPV can be diagnosed on post-mortem from a sudden death, or on exploratory laparotomy in a patient that was fully vaccinated and where CPV has been wrongly ruled-out on vaccination status alone. Here we will discuss clinical signs of CPV disease, diagnosis, treatment and prognosis.

Clinical Signs of infection

Parvovirus does not affect all dogs equally. It has been suggested that different strains may result in varied disease effects, and equally, viral dose, and virulence of the strain will play a role, as do the age of the animal, immunity, breed, and route of exposure.

CPV infection typically peaks after weaning at the age of 4-12 weeks, when maternal antibodies wane. Infection can be seen commonly in pups up to 6 months of age, and even in the 6-12 months cohort. It must also be remembered that CPV is not just a disease of puppies (though significantly more cases are young animals). CPV can occur and does occur in adult dogs, including fully vaccinated animals.

Clinical signs in some animals may be unapparent. The most common clinical signs include vomiting and diarrhoea where the diarrhea can range from mucoid to bloody. Diarrhoea does not have to be bloody to diagnose Parvo. As a consequence of the gastrointestinal damage caused by the virus, dehydration and secondary infection often develop rapidly. Pups may present at first with lethargy and inappetence but no gastrointestinal signs.

Clinically, animals may present with severe leukopenia which may be transient (or they may present with a normal WCC and leukopenia may develop during the course of the disease). Lymphopenia may be more pronounced than neutropenia and anemia can also be present but is not a consistent feature of infection. A normal WCC does not rule out CPV infection. Intestinal protein loss may occur secondary to inflammation, causing hypoalbuminaemia. Death can occur as quickly as 24 hours after the onset of clinical signs, especially in younger pups.

Puppies that are infected in utero or before 6 weeks of age may develop myocarditis due to damage as the result of viral replication in the myocardium. This is rarely seen nowadays, with one theory being that MDA protect most pups from this form of the disease.

Diagnosis

Diagnosis of CPV is commonly made based firstly on clinical signs and then confirmed by cage-side commercial testing. These tests detect antigen in fecal material and have relatively high specificity but low sensitivity. While modified-live vaccines can result in false-positive findings in
dogs 4 to 10 days after vaccination, in many cases, animals that are recently vaccinated that test positive, are in fact, clinically infected prior to vaccination. Some laboratories have data to demonstrate that vaccination will not interfere with their cage-side ELISA tests, so veterinarians should consult the provider of their tests in the event of a suspect false-positive.

False negative test results can also occur due to a lack of viral shedding at the time of testing or antibody binding to CPV antigens in faeces, and it is important for a veterinarian to consider CPV as a possible cause of gastrointestinal disease even in the event of a negative ELISA test finding. In a patient that is still deteriorating and suspected of CPV though negative at first on ELISA test, a repeat test in 24 hours may produce a positive confirmatory result.

PCR testing is also readily available and provides a highly sensitive and specific means for confirmation of CPV in patients, including for dogs suspected of parvo but testing negative at first on ELISA. Reference laboratories are now able to provide realtime-PCR testing and a Canine Diarrhoea Panel can be used to identify not only the presence of CPV, but also possible co-infectious causes of disease. Identification and adequate prompt treatment of co-infections can significantly improve the patients outcome.

In cases where exploratory laparotomy is performed or on post-mortem examination, histopathology can be used to confirm diagnosis of CPV also.

Co-infections

Dogs infected with CPV commonly carry co-infections with other pathogens which may complicate diagnosis, result in protracted clinical signs and potentially a worsened morbidity or mortality. Particularly in younger animals with already weakened immune systems from stress or malnutrition, co-infections need to be identified along with CPV, and supportive treatment needs to be provided to address both the CPV virus and other pathogens that could be causing or worsening clinical signs.

A recent worldwide study using realtime PCR demonstrated that CPV was the most common virus identified in dogs presented with gastrointestinal disease (34.6%) and was the most prevalent pathogen in the co-pathogen dual infections, associated with Clostridium perfringens alpha toxin, Cryptosporidium spp., or Giardia spp. This study also reaffirmed the finding of CPV in adult dogs (11% of cases in dogs 1-8 years and 12% of cases in dogs aged >8 years) while the disease of course remains a common cause of disease in dogs aged < 1 year of age.

Other co-pathogens that may be implicated in gastroenteritis in dogs include helminths (Ancylostoma sp. and/or Toxocara sp.), other bacteria such as Salmonella sp and E.coli sp, and other viruses such as Canine Coronavirus and Canine Distemper.

Treatment

The treatment of parvovirus infection in individual animals relies on supportive therapy and needs to be symptom-based.

Patients with CPV generally have the following issues that need to be managed:

(a) Dehydration and shock
(b) Immune suppresion
(c) Secondary bacterial infection
(d) Vomiting and nausea
(e) Gastrointestinal pain
(f) Possible endotoxic shock
(g) Potential hypoproteinaemia

Treatment therefore needs to consist of the following (parenterally rather than oral where possible):

(a) **Fluid and electrolyte therapy.** If the patient is presenting in shock, a bolus of 10–20ml/kg of a balanced electrolyte solution (e.g. lactated Ringers solution) should be given by rapid infusion (over 10 to 20 minutes). If there is no improvement in peripheral perfusion, normalising heart rate or improved demeanour then boluses can be repeated up to 60–90 ml/kg before moving onto colloid administration. Maintenance requirements (50ml/ kg/day [dogs weighing less than 5 kg may require slightly more]) and an estimation of the fluid deficit (calculated by multiplying percentage dehydration by body weight in kg) should be calculated and fluids administered to correct the deficit over a period of 6 to 24 hours, depending on its severity. Many dogs with severe gastrointestinal losses will require higher rates of fluids, e.g. 10ml/kg/hr for prolonged periods.

(b) **Colloid therapy and FFP.** In hypoalbuminaemic patients, colloidal support can be administered as a constant-rate infusion with typical rates of 10 to 20ml/kg/ day. When colloids are administered, the concurrent crystalloid is typically reduced by an equivalent rate. Fresh Frozen Plasma may also be used to provide albumin and also supplement antibodies.

(c) Potassium chloride supplementation. Ideally, electrolytes should be monitored and supplemented accordingly however, potassium chloride can be added safely at a rate of 20mmol per 1000mls fluid, even without monitoring, where vomiting and anorexia result in suspected hypokalaemia.

(d) **Glucose supplementation.** In hypoglycaemic patients, glucose can be can be supplemented to intravenous fluids, with the addition of concentrated glucose solution to make a 2.5 to 5% glucose solution, depending on the degree of hypoglycaemia.

(e) **Antiemetics.** Ongoing vomiting and nausea severely worsens patient comfort and morbidity, and antiemetic therapy is essential to patient improvement. Metoclopramide can be used as a constant-rate infusion (ideally) or as intermittent dosing, and increases g-i motility as well as reducing vomiting. Maropitant may also be used but with caution in young pups, and Odansetron is another alternative in refractory cases.

(f) **Analgesia.** CPV gastroenteritis is a painful condition and analgesia is important in providing patient comfort and improved recovery. Opioids, particularly Buprenorphine is recommended for strong analgesia with a lesser effect on g-i motility. NSAIDs should be AVOIDED due to risk in young and dehydrated patients of nephrotoxicity and g-i ulceration.

(g) **Antibiotics.** It is important to provide antimicrobial cover for CPV patients, for treatment of septic shock, due to risk of bacterial translocation due to g-i damage from the virus, because of viral immune-suppression, neutropenia, and risk of other secondary bacterial infections. Cephalosporins are recommended as primary antibiotics of choice and should be given parenterally. In severe febrile cases, potentiated amoxicillin or cephalosporin, with metronidazole provide good cover against gram negative and anaerobic bacteria that may translocate from the gut. Amikacin, gentamicin, trimethroprim sulfa and enrofloxacin can also be used for suspected secondary sepsis, however veterinarians should avoid enrofloxacin in growing pups. If using amakacin or gentamicin then pups must be well hydrated because of risk of nephrotoxicity.
(h) **Nutrition.** Recent studies have shown that early enteral nutritional therapy in dogs with CPV showed earlier clinical improvement and weight gain versus patients only fed once vomiting had completely ceased. If antiemetic therapy allows feeding without vomiting (voluntary or via feeding tube), then enteral nutrition is encouraged. Some strongly recommend feeding even if vomiting.

(i) **Interferon therapy.** Recombinant interferon (Virbagen omega, Virbac) has been demonstrated to reduce mortality and improve clinical signs and may be used as an adjunctive treatment.

**Patient Assessment and Monitoring**

Ideally, the following workup should be performed initially and ongoing monitoring should be performed on CPV patients:

(a) Full physical examination (repeat q24h)
(b) Full history
(c) Weight
(d) WCC +/- FBC (repeat as necessary, especially WCC if deteriorating or pyrexic)
(e) PCV and TSP (repeat q24 ideally)
(f) Blood Electrolytes & glucose (repeat q24h ideally)
(g) ELISA test for CPV or realtime PCR such as *Canine Diarrhoea Panel*
(h) Monitor and record appetite, vomiting, diarrhoea/faeces, urine output
(i) Faecal float or faecal analysis if necessary

**Isolation**

Isolation and quarantine of the patient to prevent transmission of disease to unvaccinated or sub-optimally protected dogs is imperative.

Where clients take home a patient that may be still shedding infectious virus (due to a mild or subclinical case, or for cost-constraint reasons were outpatient management is deemed possible) then the client must abide to strict quarantine and cleaning procedures that must be adequately explained by the veterinarian, to ensure that further disease transmission does not occur.

Patients can shed virus generally for 10-14 days post recovery (may be as prolonged as 39 days post infection) and continued isolation, and proper disposal of infectious wastes is necessary to protect others until shedding stops. If a patient is discharged when it is likely to be still shedding virus, clients should be instructed to keep the recovered patient isolated at home in a room that can be easily cleaned with diluted bleach (1 part to 24 parts water). Faeces and any contaminated items that cannot be cleaned must be double-bagged and disposed of in the rubbish collection.

**Prognosis**

Where treatment can be given promptly, and supportive therapy effective in reversing dehydration, electrolyte imbalances and protein loss, and where secondary infections and co-infections can treated, prognosis for survival with intensive care can be 90% - 95%. Many clinics report lower rates however. In untreated cases the survival rate for CPV-infected dogs may be as low as 9.1%.
Clients should be given a guarded but positive prognosis that with treatment, recovery is expected, however patients may be hospitalised for 7 or more days, and there may be a sizeable cost consideration for the intensive care required for patients to be cared for while they recover from this disease.

References:


2 Feline Vaccine Guidelines from the Advisory Panel on feline vaccines, 1998. Feline Practice, 26 (30); 14-16


