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REVIEW ARTICLE

Overview of the Parvovirus: The Traumatic Journey Ofcanine Animal

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ABSTRACT
Canine parvovirus (CPV), on the contrary end, is extremely tough, overcoming many common disinfectants and living for months to years in dirt or on microbes. This non-enveloped, single-stranded, virus named Canine parvovirus (CPV), a member of the retrovirus family, Parvoviridae which needs growing cells to replicate.Canine parvovirus variants are currently classified as CPV-2a, CPV-2b, and CPV-2c. Canine parvovirus is extremely infectious and is spread from dog to dog via feco-oral contact. It has been recorded in numerous countries. The main cause of early puppy deaths is canine parvovirus infection, a potentially lethal infectious viral disease.Mismanagement and a lack of understanding about correct vaccination schedules among pet owners are major causes of the prevalence of canine parvo virus disease in Nepal. The condition manifests itself in two forms: intestinal and cardiac, with the intestinal form being more common with hemorrhagic enteritis. The disease has been reported to be more severe in puppies than in adult dogs. Myocarditis, which is widespread in puppies, and gastro-enteritis, which is frequently seen in adults, are the two most prevalent clinical types.
Key words;*Anorexia, Lethargy,Leucopenia,Canine Parvovirus, DNA Virus, Stressors, hemorrhagic enteritis.*

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INTRODUCTION

Canine parvovirus enteritis "CPV" is an extremely infectious agent that causes gastroenteritis in a large number of puppies. CPV is a DNA virus that is not enclosed and belongs to the Parvoviridea family. The virus has a high predilection for invading lymphoid and intestinal tissue, and it is transmitted orally via faeces [1].Fever, lethargy, vomiting, dehydration and diarrhoea which alternated from mucoid to haemorrhagic are the most common recorded clinical signs associated with CPV.Suppression in leucocytes, neutrophil and lymphocytes are predicated in this condition while anaemia is a frequent finding, however it was thought to be a result of oxidative stress status rather than the virus suppresses erythropoiesis[2].The CPV-2 virus is environmentally stable and contagious for more than 6 months[3].Canine parvovirus enteritis cannot be diagnosed merely based on clinical indications since it may be confused with other disorders. The severity of clinical symptoms differs with animal age, maternal immunity, immune response status, and virus strain pathogenicity. Furthermore, certain dog breeds, such as Rottweiler and Doberman, are more susceptible to CPV-2 enteritis than others [4]. Furthermore, elements like as overpopulation, inadequate sanitation, and colony setting are related with CPV-enteritis. Infected dogs experienced acute signs and symptoms like depression, starvation, fever, vomiting, dehydration, and diarrhoea. This acute type quickly progressed to a severe subacute form with increased vomiting and profuse diarrhoea, much of which was bloody, followed by severe dehydration, decreased body temperature, collapse, and death of the infected animals[5].If the infection is not treated, the mortality rate in affected dogs may reach 100%. Even in treated situations, canine CPV infection still results in elevated moralities in infected puppies and dismal survival and recovery rates.

EPIDEMIOLOGY OF CPV

A new infection of puppies was discovered worldwide in the late 1970s. Within a year, CPV-2 was recognised as the causative agent of severe haemorrhagic gastroenteritis in dogs and quickly spread over the world. The disease was almost reported in United Kingdom, New Zealand, Canada, Belgium and Australia. In Nigeria, Canine parvovirus type 2 enteritis was reported in 1985[6].There have been questions about how Nigerian mongrel dogs are being exposed to canine parvovirus type 2. South Africa has been found to have canine parvovirus type 2 both genetically and serologically[7].Canine parvovirus types 2a and 2b are currently prevalent in varying degrees in various countries throughout the world. In Korea, canine parvovirus type 2b is the most common antigenic type. In European countries and Italy, type 2A CPV is more prevalent.

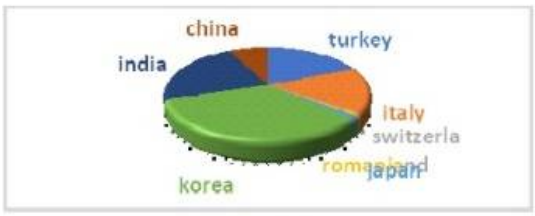

Figure 1 (a)

Figure 1 (b)

Figure 1 (c)

Figure 1 (a): Interpretation of Parvo virus Throughout World, Figure 1 (b): Interpretation of Parvo virus as per Age Group, Figure 1 (c): Interpretation of Parvo virus on basis of gender

Canine parvovirus types 2a and 2b are currently prevalent in varying degrees in various countries throughout the world. In Korea, canine parvovirus type 2b is the most common antigenic type. It is more common to find Type 2a canine parvovirus in Italy and other European nations.Among family-owned dogs, the lifetime incidence rates of CPV range from 25% to 90%[8].Because CPV-2 is exceedingly resilient and can survive in the environment for more than 6 months at room temperature, it can be carried over great distances by objects or materials that are prone to contain infection, such as clothing, cutlery, and furniture. The faeces of sick dogs are the main source of infection [9]. Reports of CPV 2 and its variants have come from a number of nations, including Turkey, Italy, Switzerland, Romania, Japan, China, India, Korea, USA, and Australia. (Figure 2).

Figure 2: The global Distribution of CPV

PATHOPHYSIOLOGY OF CANINEPARVOVIRUS

The replication occurs in the oropharynx's lymphoid tissues before reaching the circulatory system and targeting the body's rapidly dividing cells, particularly those found in the bone marrow, lymphopoietic tissues, and crypt epithelia of the jejunum and ileum, as well as cardiac cells (in young dogs) [10]. Prior to intestinal infection and gastrointestinal symptoms, early lymphatic infection is characterised by lymphopenia.Multiplication in the bone marrow and lymphopoetic tissue causes thrombocytopenia and lymphopenia, respectively, and three days after infection, rapidly proliferating intestinal crypt cells become infected, resulting in viral shedding in the faeces, which peaks when clinical symptoms appear. Necrosis of the infected intestinal crypts leads to villi collapse and loss of intestinal epithelial integrity causing haemorrhagicdiarrhoea due mainly to increased intestinal permeability and mal-assimilation from abnormal mucosal function.Normal intestinal microorganisms, such as Clostridium perfringens and Escherichia coli, can infiltrate the denuded mucosa and enter the bloodstream, causing bacteremia[11].

CLINICAL SIGN & SYMPTOMS

The clinical signs of canine parvovirus are usually the result of intestinal and bone marrow cell destruction by the virus[12].

Therefore, the sign and symptoms of the disease are associated with Anorexia, depression, vomiting, profuse haemorrehagic diarrhoea, abdominal discomfort, cardiovascular shock, dehydration, pyrexia, infection resulting from leukopenia, sudden death and congestive heart failure.

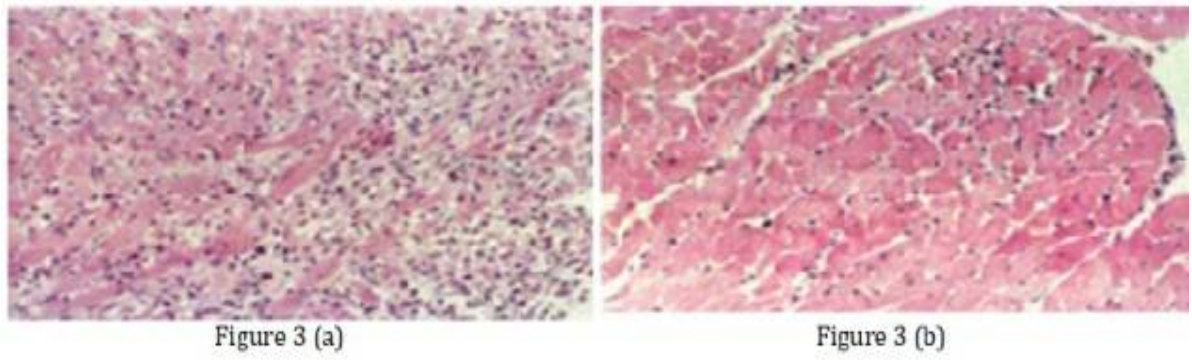

Figure 3 (a): Histopathology slides from a myocardium inflected with canine parvovirus, Figure 3 (b): Histopathology slides from a normal myocardium

Note:The presence of large numbers of inflammatory cells and the loss of pink muscle tissue in the infected section.

CLINICAL DIAGNOSIS

Canine Parvo Virus expresses clinical manifestations with parvo virus and some other virion enteritides, hemorrhagic gastroenteritis, enteric infectious diseases such as gastrointestinal foreign bodies, acute pancreatitis, hypoadrenocorticism, salmonellosis, bowel inflammation, intestinal intussusception, and numerous overdoses[13, 14].As a result, clinical diagnosis of PVE involves a combination of consistent clinical and described abnormalities, as well as the detection of the viral antigen or PCR-based replication of the viral DNA in the faeces. Canine parvovirus is diagnosed based on clinical indicators and a history of poor vaccination regimen or absence thereof, as well as confirming with a faecal antigen test (parvovirus "snap" testing)[15].

OTHER TESTS FOR THE PARVOVIRUS

Diagnostic imaging – such as radiography and ultrasound (Figure.5 & 6) can be useful in eliminating other causes of vomiting and diarrhoea, and to determine if intestinal obstruction or intussusception is present or not. It is particularly important when evaluating patients with persistent or recurrentvomiting and/or diarrhoea despite provision of intensive care over several days in hospital.

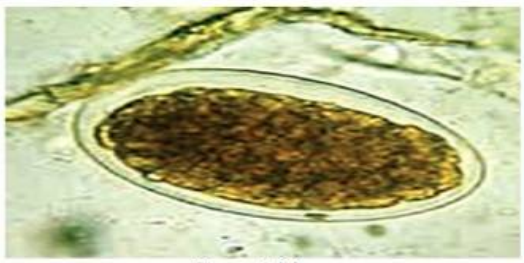

Figure 4 (a)

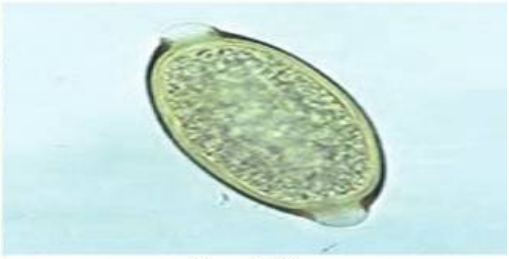

Figure 4 (b)

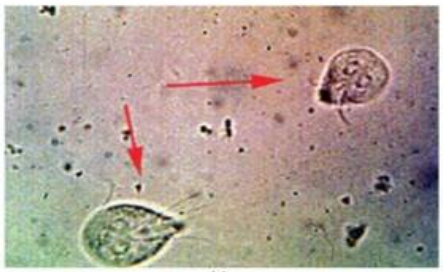

Figure 4 (c)

Figure 4 (a): Faecal analysis may reveal hookworm (ancylostoma) egg, Figure 4 (b): Faecal analysis may reveal whipworm (Trichuris) egg, Figure 4 (c): Faecal analysis may reveal Giardia cyst

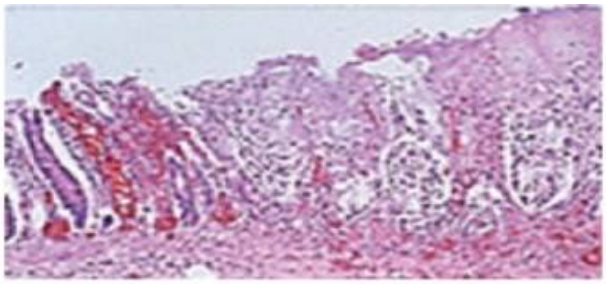

Figure 5 (a)

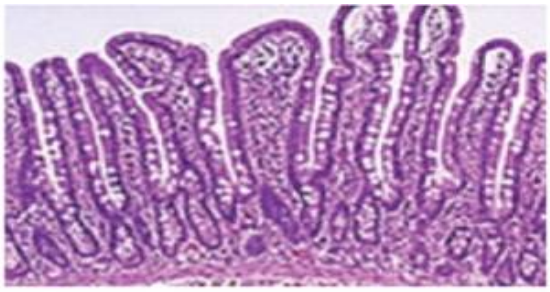

Figure 5 (b)

Figure 5 (a): Photograph showing intussusceptions, Figure 5 (b): Ultrasound image of intussusceptions

TREATMENT

Treatment for CPV is largely supportive and symptomatic. The principal components of treatment include: 1) fluid therapy, 2) antibiotic treatment, 3) antiemetic treatment, and 4) nutritional support[16].An array of other treatment measures including, though not limited to, antiviral treatments and pain management have been assessed in the past or are currently under investigation regarding their potential utility in CPV. Canine parvovirus enteritis diagnosed based on electrolyte, and metabolic imbalances and restoring fluid while also preventing subsequent bacterial infection. In the absence of significant vomiting, oral electrolyte solutions can always be administered.Colloid treatment may be a possibility if there is a significant loss of GI protein (albumin 2.0 g/dL, total protein 4.0 g/dL, indications of peripheral edoema, ascites, pleural effusion, etc.) [17]. Boluses (5 mL/kg, up to 20 mL/kg) of nonprotein colloids, such as pentastarch and hectastarch, can be administered over a minimum of 15 minutes.

Because of the potential of bacterial translocation across the damaged intestinal epithelium and the possibility of concomitant neutropenia, antibiotics are prescribed. A beta-lactam antibiotic (for instance, ampicillin or cefazolin [25-50 mg/kg, IV, every 6-8 hours]) will provide adequate coverage for gram-positive and anaerobic bacteria. For critical clinical symptoms and/or substantial neutropenia, further gram-negative coverage (eg, gentamicin [9-12 mg/kg, IV, IM, or SC, every 24 hours, enrofloxacin [5-20 mg/kg, IM, IV, or SC, every 12-24 hours]] is suggested[18-21]. Aminoglycoside antibiotics should not be given until dehydration has been treated and fluid therapy has been established. Enrofloxacin has been linked to articular cartilage injury in fast growing puppies aged 2-8 months and should be stopped if joint pain or edoema arises. Second- and third-generation cephalosporins (e.g., cefoxitin, ceftazidime,

cefovecin, and others) are also worth considering because to their broad spectrum of activity against both gram-positive and gram-negative bacteria. The drug Enrofloxacin has been linked to articular cartilage injury in fast growing puppies aged 2-8 months and should be stopped if joint pain or edoema arises. Second- and third-generation cephalosporins (e.g., cefoxitin, ceftazidime, cefovecin, and others) are also worth considering because to their broad spectrum of activity against both gram-negative and gram-positive bacteria. Antibiotic therapy is typically only needed for a short duration (eg, 5–7 days)[22-24].

If vomiting is protracted, causes symptoms and nutritional support, electrolyte imbalances, or limits oral medication and, antiemetic treatment is advised. Ondansetron (0.5 mg/kg, slow IV, once; then 0.5 mg/kg, IV infusion, for 1 hour), Maropitant (1 mg/kg, IV or SC, every 24 hours for up to 5 days) appear to be equally effective at reducing vomiting in dogs with CPV enteritis.Especially in dogs with severe stomach stasis, metoclopramide (0.2-0.5 mg/kg, IM or SC, every 6-8 hours; or 5-20 mcg/kg per hour as a constant-rate infusion) may be provided for prokinetic and antiemetic effects. Vomiting may continue despite antiemetic treatment. (Table 1).

A recent study found that faecal microbiota transplantation utilising 10 g of faeces from a healthy dog diluted in 10 mL of saline and administered rectally 6-12 hours after admission in dogs with parvovirus infection resulted in faster diarrhoea resolution and shorter hospitalisation time (median 3 days, vs 6 days with a standard therapy)[25-27].

A modified-live vaccine is advised for the treatment of CPV between the ages of 6 and 8, 10 and 12, and 14 and 16 weeks, with booster shots (Supplements) advised one year later and then every three years after that.Even though CPV may cause cerebella or cardiac cell damage, pregnant dogs or colostrums-deprived puppies should be immunised with inactivated vaccines rather than modified-live vaccines. It has been proposed that the presence of maternally acquired CPV antibodies may reduce the efficiency of immunisation in puppies aged 8-10 weeks.

Table 1; Drug Administration

Medication	Dosage	Action
Ringer Solution	Depends on the dehydration	It is used to supplement fluids and salts in the blood
Duphalyte	25 – 50 ml/5 kg	Solution made from vitamins (B1, B6, B12, Nicotinamide, Dexpanthenol), Electrolytes (Ca, Mg, Cl) and Aminoacids (Arginine, Cisteine, Anhydrous dextrose)
Tetraspan	10-20 ml/kg	It promotes retention of the fluid in the vascular system through the exertion of oncotic pressure
Metronidazole	10-15 mg/kg	The mechanism is not entirely known but it appears that in anaerobic conditions it binds to DNA and causes cell death
Pantoprazole	2mg/kg (can be divided into two doses)	Selective proton pump inhibitor, a medicine that reduces the amount of acid produced by the stomach
Hyoscine Butylbromide	1ml/10kg	It is effective against spastic pain in the gastrointestinal organs, bile and urethral tract
C Vitamin	1gm/day	It stimulates tissue oxidation processes and has antitoxic and anti-infectious action; helps maintain the integrity of the capillaries. It interferes with the healing of the lesions and the blood clotting. It increases the effectiveness of antimicrobial therapy.

Etamsylate	2ml	Improves platelet adhesion, increases capillary resistance, and reduces its permeability, Shortening bleeding time and reducing blood loss
Carbazochrome	2.5-5ml	It is capable of stopping low-intensity bleeding
Amoxicilline + Clavulanic acid	8.75 mg/kg (Once a day)	Broad Spectrum antibiotic, efficient against both Gram Negative and Gram Positive bacteria
Maropitant	1 mg/kg	Inhibits vomiting reflex by blocking NK-1 in medullary vomiting centre
Enteroguard	1 tablet/ 3 kg	Enteroguard M tablets are used to prevent and combat primary and secondary enteropathy produced by bacteria and protozoa
Eridiarom	1tablet/3 kg	Eridiarom reduces intestinal peristalsis without constipation; is astringent of the gastrointestinal mucosa, bacteriostatic, antiseptic, antihistaminic, antiparasitic and hypoglycemic.
No-Spa	2 mg/kg	Drotaverine is an antispasmodic agent whose action is based on significant inhibition of phosphodiesterase enzyme (PDE), responsible for AMP-cyclic hydrolysis (AMPc) in AMP, resulting in smooth muscle relaxation
Autohaemotherapy	5 ml of the patient	Injected in the sc axillary region

CONCLUSION

Despite the abundance of secure and extremely effective treatments, canine parvoviral enteritis is a primary reason for mortality and morbidity in dogs under the age of 6 months. Although the diagnosis is normally clear and concise (compatible clinical and haematological abnormalities in a suboptimally immunised puppy, with or without a positive faecal viral antigen test), prevention and treatment methods are constantly changing in an effort to lower the prevalence of this potentially fatal condition. Future research should aim to improve the clinical management of affected dogs by 1) enhancing monitoring tools during hospitalisation (e.g., developing more robust noninvasive markers of illness severity and prognosis), and 2) determining the optimum fluid therapy plan (eg, to substantiate the beneficial role of and refine the most effective colloid solutions), and 3) suggesting more cost-effective antiemetic and antiviral treatments. However, more study is needed to determine if the apparent vaccination failures in the clinical situation are vaccine-related (e.g., vaccinations with reduced immunogenicity against the new field variants) or vaccination policy-related (eg, level of herd immunity in an area, schedule of primary vaccination series, booster timing).

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