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Case Report

A Case Report on Suspected Parvoviral Enteritis in a Dog

Uzuegbu O. M.

Veterinary Teaching Hospital, Michael Okpara University of Agriculture, Umudike

E-mail: uzuolii@yahoo.com

Abstract

Canine parvovirus enteritis is a highly contagious serious disease of young dogs under the age of 6- 20 weeks. Infected puppies shed virus in their faeces thereby contaminating the environment and increasing the chances of infection of naive puppies. All puppies are at one point at great risk of infection even after vaccination because of period of "window of susceptibility". Therefore, dog owners should strictly observe good hygiene and disinfection of their environment and adequate exposure of contaminated formites to sunlight and heat to destroy the virus. This case in a 6 months female mixed (Alsatian and Rottweiler) breed dog recorded frank foul smelly bloody diarrhea, emaciation, anorexia, and severe vomiting. This report therefore reviewed a tentative diagnosis of parvovirus enteritis disease in dog with a view to highlight ways of management.

Key Words: Canine Parvoviral, mixed breed (Alsatian and Rottweiler) dog, management, disease.

INTRODUCTION

Canine Parvovirus (CPV) infection manifests as vomition and diarrhea in dogs below the ages of one year. It has acute and leads to high morbidity the virus types (CPV 2). It is a non enveloped, single stranded DNA virus resistant to many common detergents and disinfectants (Cynthia and Scott Line, 2010). Two pathogenic variants types (2A, 2B) have predilection for rapidly dividing cells of the gastrointestinal tract, lymphoid tissues and bone marrow, leading to hemorrhagic diarrhea, vomiting, marked leucopenia, and immune-suppression (Goddard *et al.,* 2008).

The disease is widely distributed worldwide. Antibodies to the virus exist in privately owned dogs, stray dogs and wild cannidae.

Both wild and domestic cannids are susceptible to CVP-2 infection. Young (6 weeks to 6 months) unvaccinated or incompletely vaccinated dogs are most susceptible (Cynthia and Scott Line 2010). It has been reported that Doberman Prischer, Rottweiler and German shepherd dogs appear to be more susceptible to Parvoviral enteritis than other breeds (Glickman *et al.,* 1985, Houston *et al.,* 1996).

Transmission of the infection could be by direct or indirect ingestion or exposure to feacal materials from infected animals primarily (Hassan and Hassan, 2003). The virus is shed in the faeces of infected dogs within 4-5 days of exposure (often before clinical signs develop), throughout the period of illness, and for 10days after clinical recovery (Cynthia and Scott Line 2010), the virus can remain infective for several months under good environmental condition. This is a major means of spread in canine population.

CPV-2 infection presents two forms of the disease namely, intestinal and systemic forms. CPV spreads rapidly through the faecal and oro-nasal exposure, first replicated in lymphoid tissues and then disseminates to other rapidly dividing cells notably cells of intestinal crypts, lymphoid cells, cells of thymus and those of bone marrow (Dudley and Johnny,2006).

Clinical sign of CPV infection range from asymptomatic in older and previously exposed dogs to severe fumigating signs in puppies. The clinical manifestations of CPV infection depend on the age and immune status of the animal, virulence of the virus, dose

of the virus and pre-existing or concurrent parasitic, bacterial or virus infections (McAdaragh et al., 1982, Hagiwara et al., 1996).

Initial signs may be nonspecific: lethargy, anorexia and pyrexia with progression to vomiting and haemonhagic small bowel diarrhea with 24-48 hours (Cynthia and Scott Line 2010). Consequently, there could be severe dehydration and potentially hypovolemic shock (De Laforcade *et al.,* 2003, Prittie, 2004, and Thomson and Gagnon, 1978).

De laforcade *et al.,* (2003) reported that lymphopenia and neutropenia are common leading to leucopenia often considered a hallmark of CPV infection might occur in less than 50% of the dogs at presentation.

There is usually brownish or bloody foul smelling diarrhea (Hassan and Hassan, 2003). Complications of canine Parvoviral enteritis include hypovolemic shock, electrolytes imbalance, severe metabolic acidosis, sepsis and disseminated intravascular coagulation (Prittie, 2004, Thomson and Gagnon, 1978). Other less common clinical manifestations and potential complications of the disease include acute respiratory distress syndrome, neurological symptoms and erythema multiforme (Favrot *et al.,* 2000).

Myocarditis often results in sudden death in puppies 4-8 weeks old (Hassan and Hassan, 2003). Post mortem findings in CPV infection distinct and diagnostic and include haemorrhagic enteritis in the distal duodenum. Necrosis of intestine crypts and non-suppurative myocarditis in the cardic form (Hassan and Hassan, 2003). There may be segmental discolouration of the jejunum, sub-serosal haemorrhages an congestion and thymic atrophy. The mesenteric lymphnodes may appear oedematous with multifocal pettechial haemorrhages in the cortex. Diagnosis of CPV disease is based on the history, clinical signs, laboratory tests and pathologic findings (Thomson and Gagnon, 1978). Some specific tests for definitive diagnosis of CPV include detection of viral antigen in feaces using ELISA, detection of viral particles from feces and tissues by electron microscopy and by immune histochemistry of tissue sections (Prittie, 2004). Also, indirect fluorescent antibody technique and haemagglutination test can be used to diagnose CPV.

Treatment of CPV is mostly supportive aimed at restoring fluid, electrolyte and acid base balanced, and preventing secondary bacterial infections (Prittie, 2004). Treatment include intravenous fluids and electrolytes, glucose, antibiotics, antiemeties and in cases with severe hypoproteinenmia, colloids (plasma or synthetic colloids). So the treatment is symptomatic.

Control of Parvoviral enteritis is by vaccination, but it is well known that maternal derived antibodies may interfere with the immune response of the puppies. In order to achieve appropriate protection, it is necessary to complete the vaccination schedule (Pollock and Coyne, 1993).

Case presentation

A six months old Alsatian and Rottweiler cross dog was presented with black and brown colour marking. The dog was given complete inoculation DHLPP (Biocan ®DHPPi+L, Czech Republic) against canine distemper, leptospirosis, parainfluenza, Parvovirus and Hepitis and Anti-Rabies vaccine. The dog has been vomiting since four days; it's been weak and passed out bloody smelling diarrheic faces. The dog was not eating well.
There was presence of ticks on the body, shedding of hairs, pale mucous membrane, and bilateral ocular discharge. The dog was lethargic.

Clinical examination of the dog

Clinical parameters like rectal temperature, pulse, respiratory and heart rates were respectively 41.2.^{o}C, 100 pulsations per minute, 12 breaths per minute and 108 breaths per minute. Further examination on the case revealed presence of cold extremities and reduced reflexes. On physical examination, there was presence of ticks on the body, shedding of hairs, pale mucous membrane, bilateral ocular discharges. The dog was lethargic.
The animal weighed 16kg. Based on the history and physical examination of the dog, the case was suspected for parvoviral enteritis. Blood and faecal samples were collected and sent to the Veterinary Teaching Hospital laboratory for analysis; faecal flotation and haematology.

Diagnosis

Based on the history and clinical examination of the dog, a tentative diagnosis of parvoviral enteritis was made.

Treatment

The dog was treated with ivermectn at the dose of 0.2mg/kg SC stat (10mL Liquid/Lyophilised, Polyvalent, Enzyme refined Equine Immunoglobulins, VINS Bioproducts Limited Survey, India). Aluminium Magnesium Silicate powder (Admacin$^{(R)}$) at dose of 40mg/kg PO (SAL Veterinary Service, Nigeria) for 5 days. In addition, 500 ml of 5% Dextrose Saline was administered intravenously to the dog. Metochlopromide was administered at the rate of 1mg/kg (Wuhan Grand, China); B. complex at the rate of 0.04mg/kg (Barker Alfonxo, Nigeria); Iron Dextran at the dose of 20mg/2ml (Hebei Huarun Pharmacy Co., Ltd, China) and Prazisam$^{(R)}$ Plus (Vetoquinol India Animal Health Pvt Ltd, India) at 1tablet/10kg and Paracetamol at the dose of 2mg/kg IM (Jiangsu Ruinan Qianjin, China). The laboratory examination showed mild Ancylostomiasis on

faecal flotation and there was no haemoparasite on blood smear and wet mount.

Blood count measures from a dog with suspected parvovirus enteritis at presentation at day four of hospitalization are as follows:

White blood cells (X103/µL) 4.92
Red blood cells (X106/µL) 4.06
Hemoglobin (g/dL) 12
Neutrophils (X103/µL) 8.0
Lymphocytes (X103/µL) 1.6
Monocytes (X103/µl) 1.0
Eosinophils (X103/µL) 0.3
Basophils (X103/µL) 0.00

DISCUSSION

From the clinical signs, the case is suspected to be parvoviral enteritis. This tentative diagnosis is in agreement with the report of (Hoskins, 1997, Prittie, 2004) that CPV can occur in dogs of any age though puppies (between 6 to 20 weeks) are mostly affected. Factors that predispose to parvoviral infection in puppies include lack of protective immunity, secondary intestinal parasites, overcrowding, unsanitary and stressful environmental conditions (Brunner, 1985, Smith-Carr *et al.,* 1997). In this case, detection of anclylostoma infection is in agreement as with the tentative diagnosis of CPV.

It has been reported that Doberman pinscher, Rottweiler and German shepherd are at greater risk of CPV enteritis (Glickman *et al.,* 1985, Houston *et al.,* 1996). This could possibly be due to inherited immunodeficiency in Rottweiler as well as the fact that the breeds have relatively higher prevalence of Von Willebrand's disease (Glickman *et al.,* 1985, Houston *et al.,* 1996, Prittie, 2004). This is in agreement with the case report as the dog affected is a cross of Alsatian and Rottweiler.

Clinical signs like vomiting, fever and foul smelling bloody diarrhea are in agreement with signs of parvoviral enteritis (Prittie, 2004, Thomson and Gagnon, 1978,). Therapy of 5% dextrose saline is supportive, aimed at restoring fluid and electrolyte (Prittie, 2004, Thomson and Gagnon, 1978). Treatment of CPV includes intravenous IV fluids and electrolytes, glucose, antibiotics, anti emetics and in cases with severe hypoproteinemia, colloids. Metochopromide has also been useful in treatment of dogs that have chronic vomiting. Iron dextran was given as a haematinics to boost replacement of blood loss.

Broad spectrum dewormer Prazisam$^{(R)}$ plus comprising febendazole, pyrental permeate and Praziquantel was administered to eliminate secondary helminthes infections. Antibiotics were given to eliminate secondary bacterial infections and so avoid septiceaemia. Aluminium Magnesium Silicate (AMS) has been reported to have antiviral effects (Ezeibe Maduike, 2009). All viruses are known to be electrically charged (Cann, 1993). The parvovirus is said to be negatively charged. Molecules of Aluminum Magnesium Silicate have two electrically charged ends (Ezeibe Maduike, 2009). One end has positive electrical charges while the other has negative electrical charges (Vanderbilt and Veegum, 2012). So in patients, AMS adsorbs onto viral particles released from infected cells. So, new foci of infections are minimized. Reduction of number of foci of infections gives body immune systems advantage over viral infections so that synergy between the patient's immune response and antiviral effects of the AMS could lead to recovery from the infection (Ezeibe *et al.,* 2014). This may be what led to recovery of the dog in this case.

AMS is reported to be a drug stabilizer. By this AMS protects drugs against degradation by metabolic processes. This prolongs bioavailability of the medicine and when bioavailability is prolonged effects of antimicrobials improve (Brent, 2001). So the AMS may also have improved antibacterial effects of the Ampicillin in Admacin against secondary bacterial infections. So, synergy between antiviral effects of the AMS, improved antibacterial effects of Ampicillin and enhanced immune response of the dog because of minimized foci of the viral infection may be what led to recovery of the dog. Outcome of this treatment supports results of Nwaigwe (2011) got in treating experimentally infected dogs with AMS.

CPV enteritis is an acute gastroenteritis with a more severe manifestation in puppies. Early treatment of the disease improves the prognosis. The above treatment regimen used was very effective against the disease which was evidence on day 5 of the treatment as the symptoms observed earlier have disappeared and the dog showed signs of recovery. The case report therefore agrees with the report of Ezeibe *et al.,* (2010) which showed the AMS inhibits canine parvovirus and cures infected dogs.

CONCLUSION

About 80 to 85 percent of affected dogs will survive and live normal lives if disease is detected early and proper treatment and hospitalization is sought and administered.

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