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Case Report

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Case report of canine parvoviral enteritis in 12weeks old rottweiler female puppy

Abstract

An twelve weeks old female Rottweiler puppies named Zinna was presented to Diamond veterinary center, Lagos, Nigeria with chief complains of a puppy that was normally playful and agile suddenly becomes lethargy and withdrawn to itself three days before presentation coupled with inappettance and subsequent vomiting, diarrhea within the last 24 hours prior to clinical presentation , puppy later void bloody and foul smelling feces. Puppy had temperature of 38.6°C, Pulse rate was 96 beats per minute and respiratory rate was 28 beats per minutes, the puppy had no history of previous vaccination with Canine parvo viral enteritis or canine distemper preventing vaccines. The puppy tested positive to Canine Parvoviral enteritis using commercially available ELISA test kit with puppy fecal sample , puppy was hospitalized and treat symptomically for 10 days ,puppy was discharged on day 12 and brought back for vaccination on day 16th.

Keywords: ELISA test, canine parvoviral type 2, bone marrow, lymphocytes

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Introduction

Canine Parvoviral Enteritis is an acute, highly contagious and often fatal viral enteritis of young puppies between the ages of six weeks to six months and immune-compromised whelping bitches caused by Canine Parvoviral Type -2 sub variants [CPV-2]. It was designated Canine Parvoviral Type 2 because another virus known as Canine Parvoviral Type 1[CPV-1] or Minute Canine Parvovirus has been isolated and identify previously in Germany in 1967 but this new viral isolates was found to cause a more fatal diseases in young puppies and it is antigenic ally difference from CPV-1 which lacks the pathogenic abilities and virulence associated with CPV 2 and can be found in feces of apparently normal dogs.

CPV-2 is very virulent and more pathogenic than CPV-1 and has more antigenic similarity to Feline Panleucopenia Virus [FPV] than CPV-1, in fact CPV-2 differs from [FPV] by two amino acids in it viral capsid, it is also more similar to Mink Enteritis Virus than CPV-1.

CPV-2 was first officially recognised as the caused of highly contagious new endemic fatal dog disease in North America in 1978 and later in Japan, Europe and Australia but subsequent serological retrospective studies of sick dogs serum indicates that this virus began infecting dogs in early 70s, this was due to finding of viral specific antibodies found in stored serum of sicked dogs in Greece in 1974, Netherland 1976 ,and Belgium in 1976.

In 1978 serological studies of dogs serum carried out in dogs in Japan, Newzealand, Australia and United State of America confirmed the present of the virus in those countries. Canine Parvoviral Enteritis was first reported in Nigeria in Zaria[1] and later in southern part of the country.[2]

These viruses has strong affinity for rapidly dividing cells, it first replicate in lymphoid tissues of Oral cavity and pharynx, thymus, bone marrow, mes enteric lymph nodes before it is disseminated into small intestinal crypts epithelium cells.

CPV-2 by infecting the lymphoid tissue causes immuno suppression directly through lyses of lymphocytes and indirectly through bone marrow depletion of lymphocytes progenitor stem cells inside the bone marrow. Viral replication in lymphoid tissues leads to marked atrophy of lymphoid tissues in thymus, spleenic follicles, lymph nodes of peyer patches, the same viral replication activities in epithelial cells of intestinal crypts lead to necrosis and sloughing off of intestinal.

Canine parvovirus type -2

Canine Parvovirus Type 2 [CPV2] , also know simply as PARVO, it is highly pathogenic parvovirus that affect domesticated and wild canids it is a single stranded, non envelop DNA virus, that is extremely resistant to various disinfectant but susceptible too and easily destroy by common household bleach [sodium hypochloride], it is highly mutagenic and still believe to still evolving ,it is believe to evolved from mutation from Feline Panleucopenia virus or other carnivoure parvovirus because it differ from Feline Panleucopenia virus [FPV] and Mink EnteritisVirus by only few DNA bases in its viral capsid[3,4] this might probably due to laboratory tissue culture contamination and worldwide fast distribution by vaccine contamination, however this has not been prove.

CPV -2 virion particle are small viral particles spherical in shape approximately 20nm in diameter and non enveloped.[5,6] The CPV-2 was first isolated in1978 and by 1980 it has become panzootic with new strains been isolated in 1979 designated CPV2a, this new strain has replaced the original CPV-2 virus in most infected dog isolates and it is found to be more infectious to cat than original CPV-2

There is only small antigenic variation between strains of CPV-2[CPV-2a , CPV-2b ,CPV-2c] detectable only by monoclonal antibodies and genetic analysis. CPV-2a was discovered in 1979,[7] and by 1980 has replace the original CPV2 in circulation another new strain was discovered in 1984 designated CPV 2b this strain differs from CPV2a by one or two amino acid substitution in it viral capsids 11 [VP 2].

Antigenicity

CPV-2 is closely related antigenically to FPV and MEV[8] but it has no antigenic relationship with Canine Minute Virus or CPV-1[9] and Dependo virus associated with Canine Adenovirus has minor serological cross reactivity with Swine Parvovirus, CPV 2 affect all members of canidae. Also it two clinical manifestation enteritis and myocarditis are diseases not previously seen in dog, serological survey shows that CPV-2 is new viral infection,[10] the earliest known antibodies associated with CPV-2 is the one found in dog serum in Greece in1974.

CPV 2 has high rate of nucleotides substitution similar to RNA viruses. CPV 2a and CPV2b are antigenically similar to original CPV 2 even though there are some amino acid substitution in their amino acid sequence of their viral capsid protein.

Mutagenicity

CPV-2 was first discovered in dog in North America and Europe in 1978 as a new viral disease suspected to have mutated from Feline Panleucopenia Virus or Mink Enteritis Virus by 1979 this virus has attained a pandemic status. shortly and it was reported worldwide in 1979 and by 1980 a new viral strain has evolve from it, designated CPV2a, has evolved from the intial viral strain of CPV 2 with few genetical re assortment of few viral capsid protein bases that changes it virus antigenic characteristic detectable only by deep genetic analysis and monoclonal antibodies tests. Further minor antigenic shift occur in new viral isolates in suspected outbreak in 1984 this new isolated strain was designated CPV2b.

The virus has high rate of adaption to adverse environmental conditions and high mutagenic potentials, these abilities helps in virus high rate of spreading ,it has show remarkable ability to survival in the environment under adverse condition couple with viral high rate of nucleotide substitution only comparably to RNA viruses, this abilties has help CPV2 to mutate into a new more virulent , more pathogenic ,more resistant and more stable virus with increase host range infecting abilities. CPV 2 believe to still be in it evolving stage, these abilities has continue to account for persistent parvovirus enteritis infection seen today. We now has three dominant strains that are mutant of original CPV 2 that causes disease in dog worldwide designated CPV 2a, CPV 2b, CPV2c.

Breed susceptibilty

Although all breeds of dogs are susceptible but Rottweiler, Doberman pinscher, America pitbull terrier, English springer spaniel and German sherpherd dogs are believed to has a higher risk of coming down with the disease than other breed of dog.

Epidermiology

Parvoviridae are small, non enveloped single stranded DNA viruses that are sometime species specific in causing disease in mammalian animals. Canine Parvovirus belong to the family Parvoviridae, genus Parvovirus, these are small viruses with DNA genome of about 5000 amino acids /bases with a hair pin morphology. Using X-ray crystallography it viral capsid have been found to be sixty copies of combination amino acid making up it three viral capsids designated as VP1,VP2 and VP3. VP1 has full sequence with additional N terminal domain, VP2 account for 90% of the viral capsid and it is the major determinant of host range or specificity and pathogenicity it cleaves to VP3 using host protease enzyme.

Parvoviruses has exceptional ability to evolve into a more stable, more virulent strain with increasing host range infecting ability , this has help greatly in their ability to persist in the environment couple with the fact that they can survival in the environment under unfavourable condition with large amount of viral particle shed in feces by infected dog billion of viral particles are excreted by infected dog , this active shedding can last up to 2weeks. This virus has affinity for rapidly dividing cells, this account for it tropism for lymphoid tissue, myocardium cells of puppies under three weeks, bone marrow and intestinal crypts epithelium cells of dogs Since 1981 most countries of the world has report presence of CPV 2 in their dog population but the most dominant strains isolated in Nigeria using SNAP parvo antigen test are CPV 2a and CPV2b and dominant CPV 2 strains isolates from South Africa are CPV 2a and CPV2c. It is possible we have undocumented CPV 2c strain in Nigeria because of large number illegal importation exotic dog breed from South Africa to Nigeria without adequate and proper quarantine procedure.

Most adult dogs are now resistant to this disease because they must have acquire immunity against it either by survival natural subclinical infection or through vaccination against it. Most breeding bitches are now immune against CPV2 strains and can pass maternal antibodies to their neonate via colostrum or via the placenta in the uterus. This help greatly in reducing myocardium form of the disease that is prevalent in susceptible puppies below the age of three weeks as the neonate has active maternal immunity for the first weeks of live when infection with CPV 2 can result in myocarditis[11] this make myocardium form of the disease to be rare occurrence , occuring only exclusively in pup of individual non immunized pet bitch that comes in incontact with the disease at about the time of whelping , one way this can occur is when such bitches has dystocia and they are presented for cesarean section.[12]

Although severe clinical enteritis disease occur in dog younger than six month of age, adult dog with insufficient immunity may be at risk of infection too, if they come in contact with the disease at any age.[13,14] This puppy must have had protective maternal antibodies against CPV 2 that has waned belong protective level because there were not history of the puppy been vaccinated against CPV2 at ages 7weeks and 11weeks before it came down with clinical PARVO.

Predisposing factors

Breeds: Certains breeds of dog show high risk of of coming down with CPV-2 diseases than others , reason for this increase infectivity within breed is unclear bit it has been suggested that Doberman pinscher and Rottweler breeds of dog has high risk of coming down with CPV 2 because both breed share common ancestor, they both have higher prevalence of Willebrands disease couple with the fact that Rottweiler breed are predispose to genetic immunodeficiency, also these breed are more popular and common than other breeds, lnadequate vaccine protection due to owner not following strict vaccination protocol in general. Also america pitbull terrier, Labrador retriever are also among dog breed with high risk of been susceptible to canine parvoviral enteritis[CPE] this puppy is Rottweiler breed of dog ,this breed has been know to has high risk of coming down with PARVO.

Sex: This a female puppy although CPV has been shown to affect male puppy more than female and intact male older than six month are more likely to come down with CPE than intact bitch.

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Seasonal variability: CPE is more common in summer than in winter and in Nigeria there is high prevalence of the disease from January to August , it peak occurs at February to April but no CPE are recorded in september to December.

Age incidence: Dog of any age can be infected but the incident of clinical disease is more in puppies of weaning age six weeks to six month of age , puppies younger than six weeks are protected by maternal antibodies .more adult dog are already immune due to vaccination or sero conversion immunity from sub clinical infection, after six weeks maternal antibodies concentration start dropping belong protective concentration in the serum , until about 20weeks when it deplete to such a low concentration that it cannot protect the puppy from any infection. CPE can affected stray unvaccinated dog adult dog up to one year of age.

Shelter animal: Due to exposure to many animal in close proximity confinement, puppies from shelter animal adoption centre are more likelyn to come down with the disease.

Malnutrition: Malnourished animal have high risk of coming down with CPV 2 due to low immunity. Although this puppy is on high plane of nutrition, it has access to commercially available imported puppy food.

Poor sanitation: Infected puppies can sheds infective viral particles for up to two weeks, these viral particle can survive in unsanitary favourable environment and remain infective upto eighteen month, so animal kept in poor sanitary environment were at high risk of coming down with the disease.

Transmission: CPE is transmitted directly by feco oral route and indirectly through contact with contaminated fomites , during illness sick animal continue to shed massive amount of viral antigen in feces these virion particles can survival in the environment for long time and retain their capability to be infective even long after cessation of clinical signs of disease disease, ingestion of contaminated formites from environmental contamination play a major role in transmission of CPE[12] one gram of contaminated feces from actively shedding acute infected puppy is sufficient to infect at least one million susceptible puppies by oral route.

Incubation period: Sign of enteric disease appear in 4-14 days after exposure to viral particle.

Pathogenesis: After infection by ingesting viral particles through feco oral route or through inhalation of viral particle from contaminated formites , viral replication begins in lymphoid tissues specifically lymphoid tissue of the oral cavity and pharynx, mesenteric lymph nodes and thymus and it is disseminated through hematogenous route to rapidly dividing cells of intestinal crypts epithelium cells, this last for three to five days after infection ,marked viremia developed in the plasma and it is noticed up to five days after infection.

After plasma viremia, the virus is found in many rapidly dividing epithelium cells for example epithelial lining of the tongue, esophagus, oral cavity, small intestinal crypts epithelium cells, bone marrow, spleen, thymus and various lymph nodes.

The severity of the disease is determined by cells turn over rate at these epithelial cells, higher cells turn over rate in lymphoid tissues and intestinal crypts epithelium means higher viral replication rate and more destruction of cells at these sites and more tissue necrosis observed.

During four to six weeks of age enterocytes of the intestinal crypts has higher mitotic index and higher cell dividing and replication rate, this is due to the fact that around this time there is change in puppies diet due to weaning and change in intestinal micro flora, this make puppies more susceptible to infection around this time.

Parvovirus infects the germinal epithelium of of the intestinal crypts causing destruction of the epithelium and villous damage and collapsed thus leading to characteristic pathological lesion of shortened and atrophic villi of the intestines, this altered the absorptive properties of of the intestines epithelium cells in the gastro intestinal tract.

There is extensive lymphocytolysis in the germinal centre and cortex of thymus because of higher mitotic index in the thymus, this is responsible for the lymphopenia found infected puppy. Early lymphoid tissue infection with overt clinical signs accompany by temperature raise and lymphopenia intiate the disease in all cases of clinical manifestation, there after myocardium cells and intestinal crypts epithelial cells are affected.

In neonatal puppies rapid myocytes replication occurs during the first 2 weeks of life,[15] while intestinal epithelium cells turn over rate is slow during this time[16] these situations reverse itself in the following weeks,when intestinal crypts epithelial cells start replicating actively at four weeks of age, cardiac growth continue as hypertrophy not as replication although DNA synthesis and nuclear kinesis continue until at least 8weeks of age.[15]

Infection of susceptible neonatal puppies any time as from four weeks of age result in enteritis. However infection in susceptible bitch at various stages of pregnancy does not cause intra uterine infection in fetus,[16] also Parvoviruses infection does not cause stillbirth or affect conception rate, Parvoviruses infection does not have any effect on reproduction as it does not affect incident rate of stillbirth, average litter size does not increase or reduces in an experiment conducted on two thousand brooding bitches.

Clinical forms

There are two major clinical form / manifestation of the disease that is;

Cardiac Form

Enteric Form

Cardiac form: Seen in young neonatal puppies less than three week of age and immuno compromised bitches, it manifest as sudden death in apparently normal puppy after exposure to sudden stress , excitement or exercise.

Affected puppy gasp, mucous membrane become cyanotic with death occurring under two hour of intial clinical manifestation due to non suppurative myocarditis, mortality may be up to seventy percent in affected litter. Surviving puppy from infected litter are susceptible to heart disease later in life.

By eight to twelve weeks of age surviving puppy show sign of acute heart failure [cardiomyopathy] which include dyspnoe, tachycardia, tachypnoe with ascites and hepatomegaly.[12] Sudden death is due to irregular heart beats and delay onset of chronic congestive heart failure.

Most affected puppies were infected immediately after whelping but because most bitch are now immune to CPE through natural field

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challenge or through proper vaccination protocol, there is passive transfer of maternal antibodies to puuppies thus this form of the disease is rare.[17]

Enteric form: The case under consideration presented with the Enteric form of CPE which is the most common form of the disease, CPE is the most common cause of viral enteritis in young puppies of six weeks to six months, the infection start as non specific gastrointestinal tract disturbances. Affected puppy are withdrawn, lethargy, vomiting, diarrhoea and as the disease progresses the diarrhoea become blood stinged or bloody diarrhoea, foul smelling ,this puppy manifested these classical signs although these signs are not limited to Parvoviral enteritis induces diarrhoea, animal become dehydrated , hypothermic due to diarrhoea and vomiting, in most serious and termin al cases of CPE icterus and hemorrhagic diathesis [Disseminated intravascular coagulopathy] may develop[18] but these sign are not observed in this case because the case was hospitalized and intense veterinary medical care commence immeadiately .secondary bacteria infection may lead to bacteremia and endotoxemia which in turn might lead to systemic inflamatory responses [SIR]. Death is due to dehydration and elctrolyte inbalance, leucopenia further exerbate immune system with may lead to endotoxic shock and comma.

Diagnosis

Differential diagnosis

Canine parvoviral enteritis

Canine distemper

Giardiasis

Amoebadosis

Bacterial haemorhagic enteritis

Tentative diagnosis

Canine parvoviral enteritis

Canine distemper

Laboratory examination: puppy tested positive to canine parvovirus antigen using commercially available ELISA rapid test kit product by Aptech using puppy faecal sample.

Final diagnosis

Canine parvoviral enteritis: Suspect parvoviral enteritis in young puppy of six weeks to six months with no history of proper vaccination record again CPV-2 and shows the following clinical signs, active animal suddenly withdrawn to itself, stopping eating for about two to three days, vomiting, lethargy, diarrhea. Depression and fever, this clinical signs are not specific for CPE and cannot serve as confirmatory diagnosis. Use of commercially available fecal enzyme immunoassay test [ELISA] can be use to performed rapid confirmatory diagnosis of CPE on the clinic floor which was done in this case.

Other methods of doing confirmatory diagnosis of CPE includes the following underlisted methods; Laboratory confirmatory diagnosis of CPE can be made with Haemagglutination of pig, cat and Rhesus monkey RBC at PH of 6.5 at 4°C with viral antigen from sick puppy fecal extract.

The specificity of haemagglutional is determined by titration of the sample in parallel presence of normal and immunized dog serum. Freely infected dog fecal sample contains many thousands of haemagglutinating units of viral antigens.

Electron microscopy can be used to performed confirmatory diagnosis. Viral isolation and identification from suspected sick animal fecal sample. Viral amplification of viral DNA using PCR assay of suspected fecal sample.[19]

Serology canalso be used for retrospective confirmatory diagnosis of suspected case byusing of IgM or IgG capture enzyme linked immunosorbent assay on a pair sera or use of probe based real.[20] Post mortem lesion and histopathology studies of these lesion can also aid in definitive diagnosis of CPE. There are slide agglutination test and slide inhibition test can detest all strains and genotype of CPV are commercially available using Porcine erythrocytes.

Radiography

Although radiographic examination of puppy GIT was not done , neither the less contrast radiographic image is a very good tool to arrive at confirmatory diagnosis of CPE. Contrast radiographic image of the gastrointestinal tract can detest pathological lesion in the abdominal lumen , although these changes are not specific to enteritis caused by CPE but they can aid in arrival at definitive confirmatory diagnosis, radiographic changes observed include fluid and thining of intestinal mucosa lining coupled with low intestinal motility.[21–24]

Ultrasonography

Ultrasound examination of abdomen can detest abdominal and peritoneal effusion and intussusception. Ultrasound examination was not performed before this puppy was discharge although, it is a very good way to diagnose CPE.

Clinical pathology

Prominent histological examination finding of complete blood cells in CPE cases include leucopenia due to neutropenia and lymphopenia as a result of destruction of bone marrow precursor stem cells due to viral replication activities lymphopenia is due to depletion of stem cells destruction of lymphoid tissue parenchyma and lysis of lymphocytes. Leucopenia is so severe that leucocytes count could be as low as 500- 2000 leucocyte per microlitre or less, more leucocytes count or rebound neutrophilia is useful indicator of recovery in sick animal. This was done in this case and the result listed (Table 1).

Table 1 Prominent histological examination finding of complete blood cells

Leucocyte		
Total WBC(/ul)	3250	
Differential	%	Absolute
Seg Neutrophil	49	1592
Band Neutrophil	4	130
Lymphocyte		
Monocyte	37	1203
Eosinophils	6	195
Basophils	4	130
Nucleated RBC	0	0

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Haematocrit

it can be variable, not specific good indicator, it can be low due to intestinal hemorrhage or high due to dehydration from fluid loss as result of vomiting and diarrhea (Table 2).

Table 2 Haematocrit value for this puppy under consideration

Erythrocyte		
Hb(g/dl)	10	
PCV%	31	
RBC(106/udl)	5.27	
MCV( )	58	
MCHC(g/dl)	32	
Reticulocyte%	0	
RDW		
Platelet (/ul)	50,000	
MPV		
Leucocyte		
Total WBC(/ul)	3250	
Differential	%	Absolute
Seg.Neutrophil	49	1592
Band Neutrophil	4	130
Lymphocyte		
Monocyte	37	1203
Eosinophils	6	195
Basophils	4	130
Nucleated RBC	0	0

Serum chemistry

Serum chemistry is not a good specific indicator of CPE because result obtained can be seen in other enteric cases. Noticeable hypokalemia is due to anorexia, vomiting and diarrhea, hypocalcemia is due to hypoalbuminemia with may be relative hypoalbuminemia or absolute hypoalbuminemia with might be due to reduction in plasma protein concentration due to intestinal hemorrhage or hemodilution dilution due to fluid re hydration therapy, there is noticeable increase in alpha 2 globuline concentration despise reduction in plasma protein this can be due to hepatic synthesis of acute phase protein [APP] stimulated by endogenous leukocytes mediator that are produced as a result of tissue damage and inflammatory process, production of acute phase protein lead to reduction in albumin synthesis, there is increase in Alkaline phosphatase and Alanine transaminase as a result of reduce oxygen concentration in the liver due to low circulating blood delivered to the liver or due to many circulating bacteria endotoxin as a result of compromised intestinal epithelial absorption capacity due to destruction og GIT lumen, PH can be acidic or alkaline depending or predominant ion loss due to vomiting or diarrhea, [vomiting lead to loss of hydrogen ion and chloride ion loss, while diarrhea ion loss depend on the origin of the diarrhea it is small intestine or large intestine] majority of CPE cases show metabolic acidosis due to excessive loss of bicarbonate ion[HCO_3], unlike in human total ionized magnesium concentration can not be use as a good indicator good prognosis (Table 3).

Table 3 Serum chemistry result for the puppy

Serum chemistry		
Analyte	**Unit**	**Sample**
Total Protein	g/dl	5.8
Albumin	g/dl	2.1
Globulin	g/dl	3.7
AST	ul	16
ALT	ul	104
ALT	ul	117
GDT		
SDH		
GGT		
BUN	mg/dl	34
Creatinine	mg/dl	1.7
Plasma		
Colour	Normal	
Other observation		
Total protein	(g/dl)	6
Fibrinogen	(mg/dl)	100

Serology

Determination of positive antibodies against CPV can be misleading because 95% of dog population now have sero convertion due to previous exposure to CPV in sub clinical infection in the environment or through vaccination so they will test positive. Specific serology test for IgM analysis by indirect fluorescent antibody [IFA] or Mecaptoethanol procedure provide more definitive serological evident of recent infection because IgM is only found in first week of clinical infection.

Positive definitive confirmatory diagnosis of Canine Parvoviral Enteritis required demonstration of active secretion of viral antigen in feces which can be done on site by [ITE-parvotest, IDEX, Assure parvovirus symbiotic] all these are commercially available ELISA test ,easily to conduct and give reliable positive result which indicate active fecal excretion however recent vaccination with attenuated live vaccine may give similar result too. CPE was confirmed in this case using commercially available ELISA test kit produced by APTECH.

Management

Chances of survival for clinically infected puppies increases if

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such puppies are place on intense veterinary medical hospitalization and clinical abbe ration signs and symptoms are managed as soon as they are observed.

Although CPE start with non specific enteritis clinical signs, CPE should be suspected in any young puppies of six weeks to 6months with or without proper vaccination history coming down with any signs of enteritis disease, the treatment should commence as soon as possible, infected puppies has been chances of survival if they were placed on appropriate par enteral fluid therapy to manage electrolytes inbalance and dehydration due to vomiting and diarrhea couple with bactericidal broad spectrum antibiotic par enteral injections.

Fluid therapy

one of the major noticeable clinical signs of CPE is intractable vomiting and projectile foul smelling diarrhea, these clinical signs cause rapid electrolytes in-balance and depletion of electrolyte ion distorting the animal acid based balance and normal body internal homeostasis. Replacement and maintenance loss body electrolytes is one the major cardinal point of successful CPE management. Determination of appropriate crystalloids to use in replacement of loss electrolytes is very important because in CPE both metabolic acidosis and metabolic alkalosis can be observed, determination of body PH can be done or use of isotonic crystalloids like normal saline and lactated Ringer, Ringer lactate solution should be use with cautions in cases of CPE with severe metabolic acidosis or metabolic alkalosis or if there is any indication extensive hepatic damage that might effect lactate metabolism or when administering ceftriazone as systemic antibiotic in CPE management.

Also any puppies with noticeable hypersensitive reaction to cereal product should not be given any crystalloid that contains dextrose, metronidazole infusion should not be given with lactate ringer infusion over a long period of time.

Intravenous routes is most preferred route of fluid administration because severe dehydration impaired absorption of fluid from subcutaneous routes, intravenous routes also help to rapidly replace and correct electrolytes in balance in circulation in puppies with hypovolemic shock colloidal fluid can also be administered with 50% isotonic crystalloid fluid to help improve circulation oncotic pressure loss due to high protein loss pottassium chloride at dose rate of 20 mEq/l is administered with fluid to help correct hypokalemia normally observe in CPE. Fluid replaced at a dose rate of 40-60ml /kg body weight multiple by percentage deficit.

Antibiotic

Par enteral administration of broad spectrum antibiotic that is bactericidal is essential in CPE management because of disruption of intestinal mucosal integrity with adversely affect intestinal normal micro flora population aminoglycosides are very effective in well hydrated puppy. Cephalosporin are very good but concomittant administration of ceftrizone with lactate Ringers solution should be avoided to avoid calcium precipitation.

Antiemetics

Anti-emetics are very important set of drug use in CPE management because of frequent vomiting, metoclopramide is a dopaminergic antagonist that block chemo receptor trigger zone and also has prokinetic effect on GIT very effect but strongly contraindicated in CPE puppies with accompany intussusception.

Ondasetron and dolastron both serotonin receptornantagonist can also be use in case of freqent uncontrollable vomitting, metoclopramide, ondansetron and dolasetron are antiemetic that can act centrally and peripherally to stop nausea and vomitting caused by central and peripheral stimulation of vomiting pathway. The antiemetic we use in managing this case was metoclopramide intramuscular injection.

Nutritional support

introduction of bland enteral feeding early has improve chances of puppies survival and improve earlier restoration of mucosal integrity faster.

Pain therapy

Colic as a result of hemorrhagic enteritis and intestinal intussusception is a common sign in CPE use of analgesic like Butorphenolor Buprenorphine are beneficial or Hyoscine butylbromide [Buscopan] an anticholinergic drug is also very good in reduce abdominal pain observed in CPE.

This case was managed as described below;

puppy was rehydrated with 600ml of Darrow CRYSTALLOIDS solution in divided dose of 200ml with 100ml intravenous infusion of 500mg metronidazole over period of 48 hours and was placed on gentamycin 5mg \kg iv for first 4 days and Gentamycin 5mg\kg im for remaining 6days before it was discharged. Metronidazole infusion of 100ml daily for three day

Give oral rehydration solution ad libum

Metocloprpaomide injection i\m for 5day

Mist kaolin per os

Puppy was on admission for ten days

Start responding to treatment on day four [start notice clinical improvement on day 3]

Finally discharge on day 12

Calculation of replacement fluid =body weight *percentage dehydration *1000=6*.1*1000=600ml

Prevention

Canine Parvoviral Enteritis can be effectively prevented by following strict vaccination protocol in susceptible pet population by using polyvalent vaccines containing antigen for canine distemper, canine hepatitis, leptospirosis, CPE and canine parain fluenza. These vaccine contain modified live strains of CPE and can be administer at 6-8weeks, follow by first booster at 10-12 weeks and second boosting shot can be given at 14-16 weeks old they repeat at 6 months to 12 months later. This schedule is endorsed by world small animal veterinary association.[25]

Most commercially available vaccines contains modified live vaccines that can be use to prevent infection in susceptible animal or protect already infected puppy, some of these vaccine can provide immunity cover that can last up to 5-7years. Any puppy that succumb to infection after completing the intial vaccination protocol at 16 weeks should be re vaccination twice at four week interval. In shelter environment or overcrowding population puppy can start receiving CPE Vaccine at four week old and repeat after 3-4 weeks later good

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hygiene and strict bio security sanitary protocol are very important in limiting outbreak of CPE infection in susceptible population

wash all contaminate hard surface and formite with disinfectant with sodium hypochloride is very effective in killing the virus.

How to improve prognosis of clinical cases of canine parvoviral enteritis

Prompt commencement of treatment of suspected cases

Hospitalization of sick dog

Constant rehydration of loss fluid through vomiting and diarrhea

Good antibiotic treatment regime

Previously vaccinated dog has better chances of survival

Animal with previous acquired immunity through maternal antibiotic has good chances of survival

Good sanitation

Control

Proper vaccination schedule

Intense hospitalization of sick dog

Good nutrition

Good hygiene

Reduce overcrowding

Conclusion

Canine Parvovirus Enteritis although very contagious and lethal viral enteritis can be prevented by vaccination and good hygiene, also if susceptible puppy succumb, hospitalization of such animal greatly improve their chance of survival.

Highlights

Canine Parvoviral Enteritis (CPE) is highly contagious and relatively common cause acute viral gastroenteritis disease of young dogs and other wild canidae. Canine Parvoviral (CPV) can cause acute infectious gastroenteritis illness in young dog of 6weeks to 7 months, it is usually very fatal in unvaccinated puppy, incomplete vaccinated puppy or dog without maternal immunity or unvaccinated puppy with waned maternal immunity.

Etiology: single stranded DNA virus, non-enveloped belonging to genus adenovirus , has 2 pathogenic strains CPV 1 AND CPV 2 , CPV 2 has three virulent pathogenic sub strain i.e CPV 2a, CPV2b, and CPV2c.

Virus is highly mutagenic

Most of these strains are cover by vaccines.

The disease can spread from dog to dog through direct or indirect contact

This disease is common in Rottweiler, German sherpherd, Doberman PINSCHER, America pitbull terrier breed of dog (merck veterinary manual).

Vaccination can prevent infection mortality rate can be up to 91% in untreated cases. CPV does not infect human(National centre for immunization ,respiratory diseases). Other susceptible animal includes foxes, wolves cat and skunt. CPV can be diagnose through detection of CPV2 antigen in feaces of infected dog using ELISA or hemagglutination test ,PCR OR electron microscopy. If the infection is not that much viral been shed in the faece, ELISA may fail to detect it (intensive care treatment of severe parvoviral enteritis).[26] Cardiac form is more acute and easier to diagnose because of distinct clinical signs.[27]

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None.

Conflicts of interest

The authors declare there are no conflicts of interest.

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