Concurrent infection with canine parvovirus and *Toxocara canis* in a puppy: a case report

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Abstract

This case report describes a concurrent infection with canine parvovirus (CPV) and *Toxocara canis* in a two-month-old male Japanese Spitz crossbred puppy presented to the Veterinary Teaching Hospital of Nepal Polytechnic Institute. The puppy showed severe clinical signs including anorexia, vomiting, mucous-stained hemorrhagic diarrhea, and marked depression. Fecal examination revealed the presence of *Toxocara* eggs, while CPV infection was confirmed through clinical and pathological findings. Post-mortem examination demonstrated extensive gross lesions, including hepatic congestion, pulmonary congestion with frothy exudates, linear intestinal hemorrhages in the colon, and adult *T. canis* worms in the small intestine. Histopathological examination revealed interstitial pneumonia with edema, viral enteritis characterized by villous atrophy, congestion of the liver and lungs, and evidence of larval migration associated with inflammation and necrosis. This case highlights the synergistic pathological impact of parasitic and viral coinfections in young puppies and underscores the importance of early diagnosis, routine deworming, and prompt supportive therapy to reduce morbidity and mortality.

Keywords: Canine parvovirus; *Toxocara canis*; Coinfection; Puppy; Histopathology; Post-mortem examination

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Introduction

Canine parvovirus, also known as canine panleukopenia is classified under the Protoparvovirus genus and Parvoviridae family (Fazila *et al*., 2020). It is an infectious DNA virus that commonly causes acute gastrointestinal illness in young and unvaccinated dogs. It damages the various lining of the guts, resulting in severe hemorrhagic diarrhea, pyrexia, vomiting, and dehydration, whereas the myocarditis form leads to myocarditis in utero or after birth, showing symptoms before the age of 3-month-old. It also attacks infection-fighting cells within the bone marrow, which weakens the immune system. Predisposing factor to parvovirus infection includes intestinal parasites, inadequate protective immunity, sanitary compromises, overcrowding, and stressful conditions (Moore, 1983). Viral replication takes place in rapidly dividing cells such as precursor cells in the bone marrow, intestinal crypt epithelial cells, and myocardiocytes (MacCartney *et al*., 1984; McCaw and Hoskins, 2006; Goddard, 2010). This virus is highly contagious and spreads through direct contact with an infected dog or by indirect contact with a contaminated object.

Toxocara canis (*T. canis*, also known as dog roundworm) is zoonotic helminth parasite that primarily infects dogs and other canines. Dogs become infected when they ingest infective eggs from the environment, raw meat or prey on an infected paratenic host (e.g. rodents). Puppies are more susceptible to roundworms because their immune systems are not fully developed. *Toxocara* infections are commonly diagnosed by examining the feaces with centrifugation flotation methods. *Toxocara canis* is highly reported in young (< 1 year of age), stray, rural, male dogs, that live in tropical countries with a low Gross Domestic Product (Hotez and Wilkins, 2009; Rostami *et al*., 2020). The dog living in this region susceptible for *Toxocara* infection, as the conditions they live in are ideal for the propagation of this helminth. The environmental conditions favorable to the parasites and the large number of dogs (generally not dewormed) born due to haphazard breeding of dogs have led to increased prevalence in this region.

Moving into the pathogenesis of *Toxocara canis* in dog, Puppies are born with prepatent worms and egg expulsion has been reported to start around day 16 postpartum (Lloyd *et al*., 1983). Vertical transmission continues postpartum as larvae can be passed lactogenically to the newborn pups with a prepatent period (PPP) of 28 days (Schwartz *et al*., 2022) Horizontal transmission is facilitated through ingestion of embryonated eggs with a PPP of 32–35 days (Dubey, 1978) or through ingestion of paratenic hosts with a PPP of 34–48 days (Manhardt and Stoye, 2010). After young dogs ingest embryonated eggs, L3 larvae are released from the egg, invade the gut wall, and undergo hepatic-tracheal migration ending up in the small intestine where they mature into adult worms (Roberts *et al*., 2013).

As canine parvovirus replicates only in dividing cells, so any concomitants infestation like parasitic, bacterial or viral infection can predispose animal to CPV. There is no any report currently from Nepal which describes the concomitants infection with *parvovirus* and *Toxocara canis*. The purpose of this study was to describe the clinico-pathological characteristics of concomitant natural infection of *Parvovirus* with *Toxocara canis* in a puppy dog.

Materials and methods

Animal and samplings

A two-month-old, 4-kg intact male Japanese spitz cross (mimi) was brought to Nepal Polytechnic Institute, Veterinary Teaching Hospital with dull and depressed demeanor, anorexic, vomiting and mucous stained bloody diarrhea since last 5 days. The puppy was in moribund condition with heavy flea's infestation. Mimi had not received vaccinations against Canine Parvovirus (CPV) and had not undergone deworming treatments. The feces and blood samples were collected for clinicopathological examination. The feacal sample was examined by feacal floatation methods immediately after collection. The feaces was tested for antigen against parvovirus with parvofecal antigen test kit. (Take and Test) following the manufacture instruction. The blood sample was collected with needle and syringe and preserved in K3 EDTA tube. The blood smear was immediately prepared and stained with Giemsa stain. The dog died during treatment. The necropsy was done and gross lesions were recorded. The tissue samples (lungs, liver intestines and heart) were collected in 10% neutral buffered formalin for gross and histopathological examination.

Clinicopathological investigation

The physical examination was done and recorded. The blood smear analysis was done for estimation of platelet and leucocyte number by the methods described by (Tvedten *et al*., 1988). The feaces was collected and tested for parvovirus antigen by applying the Antigen Rapid CPV Ag Test Kit which is a chromatographic immunoassay for the qualitative detection of Canine Parvovirus antigen in canine feces. The feaces was tested for parasites by applying direct smear and fecal floatation test.

Results

Clinical and clinicopathological findings

The puppy presents with symptoms of tachycardia (172 beats/min), irregular pulse, dyspnea by tachypnea at 47 breaths/min, pale mucous membranes, prolonged capillary refill time (>2sec), poor body condition with ribs and vertebral prominence visible, tucked up abdomen, lean body mass, rough hair coat, shrunken and droopy eyebrows, hypothermia, and cold to touch, accompanied by dehydration approximately 12% (Table 1), with tented skin, sunken eyes, and dry mucous membranes, along with widespread flea infestation. The blood

smear analysis revealed a reduction in leucocytes, neutrophils, and thrombocytopenia, alongside rouleaux formation. Subsequently, fecal floatation and direct smear analysis revealed the presence of Toxocara eggs (Figs. 1 and 2). Rapid Diagnostic Test (RDT) utilizing the Antigen Rapid CPV Ag Test Kit returned a positive result.

Table 1: Physical findings in dehydration.

Percent dehydration	Clinical sign
	No detectable
<5	Subtle loss of skin elasticity, definite delay in return of skin in normal position and eye possibly sunken in orbit.
6-8	Possibly dry mucous membranes, tented skin stands in place, eye sunken in orbit.
10-12	Dry mucous membrane
12-15	As above, plus sign of shock (tachycardia, cool extremities, rapid and weak pulses, prolongation of CRT). Death may be imminent.

(Source- Rebecca Robinson VT46.35 | September 05, 2016) vet. Times)

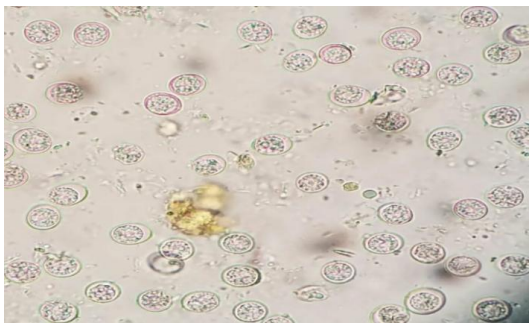

Figure 1 : *Toxocara* ova in fecal sample.

Figure 2 : Recovered adult *Toxocara canis* worm from feces.

Diagnosis

On the basis of history, physical, clinical and laboratory test the case was presumptively diagnosed as Concomitant infection of a Parvovirus with *Toxocara canis.*

Treatment approach

Based upon condition of animal immediate treatment was started focusing on severe dehydration and shock condition of animal. Peripheral intravenous access was attempted but, due to Mimi blood volume depleted state, multiple attempts at cephalic catheterization was failed. Then Jugular vein was accessed for fluid therapy. Based on hematological and physical examination hypovolemic and distributive shock was suspected and balanced isotonic crystalloid solution (ringer lactate) was chosen (Silverstein, 2011), 160 mL shock bolus of fluid (1/3rd of shock bolus 90 mL/kg) was given and monitored regularly. Dehydration was estimated to be of 12% according to Table 1 and calculated as follows

The estimated dehydration was determined to be 12%, resulting in a fluid deficit of 480 ml based on a body weight of 4 kg. This deficit was partially

corrected with a 120 mL bolus fluid volume, leaving a remaining deficit of 360 mL. To address this, fluids were administered at a rate of 30 mL per hour over a 12-hour period. Additionally, ongoing losses due to vomiting and diarrhea were estimated at 75 mL every 24 hours, equivalent to 3.1 mL per hour. Considering maintenance fluid requirements (Leah A. Cohn 2015), 240 mL was determined to be necessary every 24 hours, resulting in an hourly rate of 10 mL. Thus, a comprehensive fluid management plan was devised to address both the initial deficit and ongoing losses while ensuring adequate hydration and electrolyte balance. Ampicillin and cloxacillin (25mg/kg of bwt.) was given intravenously. After giving intravenous fluid therapy, the animal recovered and came to normal from shock state. Animal was admitted to hospital without fluid therapy and animal died in the morning. A necropsy was performed, during which gross and microscopic lesions were recorded for further analysis (Table 2).

Table 2: Maintenance fluid requirements for poppies.

Table	**Maintenance fluid requirements for poppies**
Age	Fluid requirements
Neonates (0-2 weeks)	120-180 ml/kg Q 24 hrs.
Pediatric patients (3-6 weeks)	80-100 ml/kg Q 24 hrs.
Puppies (7 weeks – 1 year of age)	60 ml/kg Q 24 hrs.

Source- Leah A. Cohn, todays veterinary practice. Oct 2015

Post-mortem examination

The liver appears highly congested and dark in color, while the duodenum contains scanty dark brown feces (Fig. 3b). The lungs exhibit rib impressions with alternating areas of pale and red color on the surface. Additionally, the heart is mildly dilated with pale streak areas present (Fig. 3c). Linear hemorrhagic necrotic areas are visible in the colon (Fig. 3d). The small intestine, though empty, contains yellow to brown mucous fibrinous exudates and adult toxocara parasites (Fig. 2 and 3e). Furthermore, the mesentery and serosal surface of the intestine were congested (Fig. 4A).

Histopathological examination

Severe congestion is observed in the liver (Fig. 4A), with centrilobular areas showing dilated sinusoids filled with blood and mild fatty degeneration of hepatocytes (Fig. 4B). Additionally, there is mononuclear cell infiltration in portal areas indicative of cholangitis (Fig. 4C). The lungs exhibit severe congestion along with thickening of the pleura (Fig. 4D), and intense alveolar septal thickening associated with diffuse chronic interstitial pneumonitis (Fig. 4E). In the intestine, mild desquamation of epithelial layers of the small intestine is noted (Fig. 4F), accompanied by infiltration of inflammatory cells indicating enteritis (Fig. 4G). Furthermore, the heart displays epicardial congestion and edema (Fig. 4H), with edematous and congested myocardium (Fig. 4I).

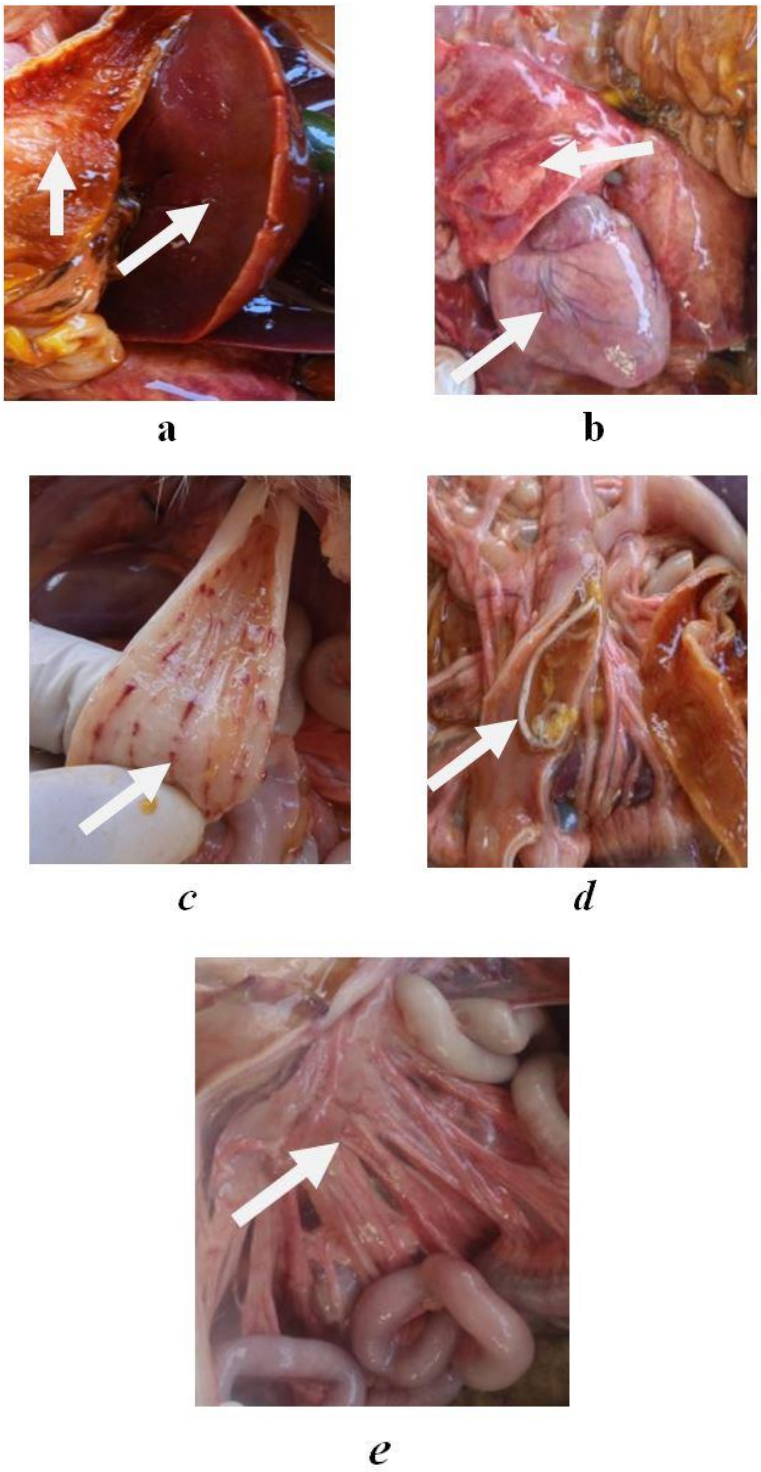

Figure 3: a) Liver highly congested and dark in color, Duodenum- scanty dark brown feces, B) Rib impression with pale and red color alternating area on lung surface, Edematous and witty lungs. Heart is mildly dilated and having pale streak area, c) Colon having linear hemorrhagic necrotic area, d) Small intestine was empty but filled with yellow to brown mucous Fibrinous exudates. Some adult Toxocara parasites are also seen, e) Mesentery and serosal surface of intestine are congested.

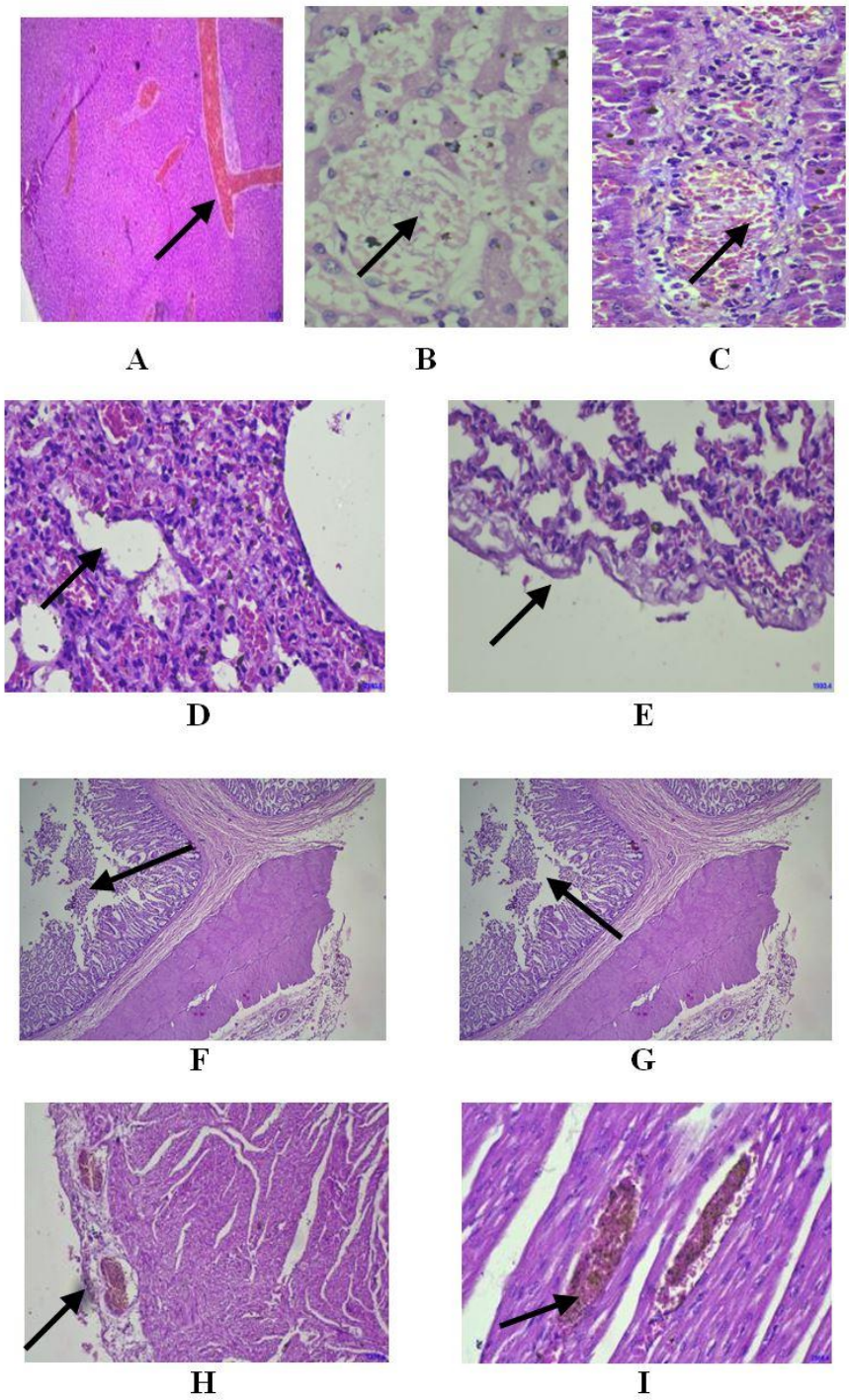

Figure 4: A) Liver: Severe congestion, B) Dilated sinusoids filled with blood fatty degeneration in hepatocyte, C) Mononuclear cells infiltration in portal areas (cholangitis), D) Lungs: Severe congestion, Pleural thickening, E) Intense alveolar septal thickening associated with intense and diffuse chronic interstitial pneumonitis, F) Intestine: Mild desquamation of epithelial layer of small intestine, G) Infiltration of inflammatory cells in epithelial layer showing enteritis, H) Heart: Epicardiuam congestion and edematous, I) Myocardium is edematous and congested.

Discussion

The dog included in this study had clinical signs and had clinicopathologic abnormalities compatible with concomitant canine parvo virus (CPV) and *Toxocara canis* infection. The presence of intestinal parasitism as predisposing or aggravating factor for CPV infection is reported in the literature (Lutz, 1919; Silva *et al*., 2021). The diagnosis of CPV in this study was done by rapid CPV Ag Test Kit in feaces. In one study done by (Kantere *et al*., 2015), revealed that snesitivity of in clinic rapid test was very low but specificity was 100%. they concluded that negative results do not exclude parvoenteritis from the differential diagnosis, especially in dogs with early vaccination history, but a positive result almost certainly indicates CPV infection. But correct diagnosis is based on a combination of history, clinical signs, biochemical parameters and positive fecal results. Aslo in this study clinicopathological findings are compatible with CPV. parvo virus (CPV). Canine *parvo virus* and *Toxocara canis* is a common and severe pathogen that affects young dog that are unvaccinated, under-vaccinated. Loss of both intestinal epithelial villous and crypt cells leads to malabsorption and increased intestinal permeability, accompanied by vomiting, diarrhea and GI bleeding. Destruction of bone marrow cells results in leukopenia and neutropenia and, to a lesser degree, thrombocytopenia which is seen in this case. CPV infected dogs exhibited clinical signs such as anorexia, vomiting, dehydration, bloody diarrhea and paleness of mucosa. Dongre *et al*. (2015) showed that initiation of therapeutic process with fluid and antibiotic cause significant improvements in first 4 days in infected dogs with *parvo virus* and dog retured to normal life in 6-8 days. The results of the present case showed similar improvement after fluid therapy. Munibullah *et al*. (2017) and Singh *et al*. (2008) also reported similar results. The highest rate of CPV occurrence in young pups may be due to viral attraction for rapid multiplying intestinal crypt cells with the highest mitotic catalogue due to alternation in bacterial flora (Deka *et al*., 2013). CPV can be controlled by providing good nutrition, hygiene environment, reduce overcrowd and vaccinate the dogs timely (Odueko, 2019). Without treatment, CPV can be life threatening due to sepsis, severe fluid losses and electrolyte dearangements secondary to anorexia, vomiting and diarrhea. In order to ensure the best outcome, treatment should be aimed towards symptomatic supportive care, aggressive fluid therapy, antiemetic, antibiotics therapy, and nutritional support.

Gross lesions observed in this study are nonspecific. Haemorrhagic gastrointeritis and necrotic lesion observed in the gastrointesinal tract is compatable with CPV (Atalay Vural and Alcigir, 2011) and *Toxocara canis*. Histopathological lesions like congestion, fatty changes, cholangitis and necrosis in liver and intense alveolar septal thickening associated with diffuse

chronic interstitial pneumonitis in lungs seen in this study is very much compatible with toxocariasis (Klockiewicz *et al*., 2019). Congestion and edema seen in different organs are nonspecific. Canine Parvovirus (CPV) can lead to myocarditis depending upon timing of infection. When the infection occurs either in utero or within the first few weeks of a dog's life, the virus aggressively targets rapidly dividing cells throughout the body, including myocardiocytes (Prittie and Barton., 2004; Sime *et al*., 2015). The virus also infects the intestinal epithelium, resulting in crypt necrosis, crypt dilatation and villous atrophy, which is diagnostic of the disease (Cooper *et al*., 1979). The most prevalent enteric form of CPV targets lymphoid tissues in the oropharynx, mesenteric lymph nodes, and or the thymus. Subsequently, the virus spreads throughout the body via the bloodstream, ultimately leading to the destruction of intestinal villi (Goddard, 2010). Given the short intestinal epithelial cell turnover time of 1–3 days, recovery is possible with intensive supportive care even with severe villus damage (Prittie, 2004). Death, however, can occur secondary to severe dehydration, hypovolemia from marked gastrointestinal fluid and protein loss and sepsis from bacterial translocation and leukopenia. All these clinical manifestations were observed in the examined puppy. It appears that younger puppies are more susceptible to CPV infection, as evidenced by the incidence observed in the report and findings by (Ezeokoli *et al*., 1985) and (Fagbohun and Omobowale, 2018). This suggests that age plays a significant role in the severity and outcome of CPV infection, emphasizing the importance of timely and appropriate intervention, especially in younger canine populations. All these physiological alterations generate a high mortality rate (Mylonakis *et al*., 2016). Parenteral administration of wide-spectrum bactericidal antibiotics (such as ampicillin) is warranted in dogs with severe CPV due to the high risk of septicemia associated with the disruption of the mucosal barrier and the concurrent profound neutropenia.

It is common to detect hematological alterations in CPV-2 infected puppies, which are characterized by a reduction of red blood cells and leukocytes, such as neutrophils and lymphocytes (Mylonakis *et al*., 2016). These events may be associated with thymus atrophy, gastroenterological bleeding losses, hyper inflammation, and CPV-2 tropism towards the bone marrow precursors (Decaro and Buonavoglia, 2012).

Conclusion

Canine Parvovirus is an infective and highly contagious viral disease of dogs. Dogs of all age groups are infected, but puppies in the age less than 3 months were more susceptible than adults. The rate of the infection was higher in non-vaccinated than vaccinated dogs. Definitive diagnosis is done by molecular methods like PCR but presumptive diagnosis with nearly high accuracy can be done with typical clinical signs and rapid diagnostic kit.

Specific treatment is unknown but with help of resuscitative fluid the pay and vigorous supportive treatment, mortality can be reduced to high level. However, control and prevention of parvovirus infection by vaccination would be best option.

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