RESEARCH ARTICLE
Open Access

Journal of Veterinary and Animal Sciences
ISSN (Print): 0971-0701, (Online): 2582-0605
https://doi.org/10.51966/jvas.2023.54.1.71-78

Canine parvo viral enteritis in dogs: Diagnostic and therapeutic evaluation#

G. Shruti [1]* and K. Ajay[2]
Department of Veterinary Medicine, Dr. G.C. Negi College of Veterinary and Animal Sciences, Chaudhary Sarwan Kumar Himachal Pradesh Krishi Vishvavidyalaya, Palampur, Himachal Pradesh, India.

Citation: Shruti,G. and Ajay,K. 2023. Canine parvo viral enteritis in dogs: Diagnostic and therapeutic evaluation. *J. Vet. Anim. Sci.* **54**(1):71-78
DOI: https://doi.org/10.51966/jvas.2023.54.1.71-78

Received: 04.08.2022 *Accepted: 25.10.2022* *Published: 31.03.2023*

Abstract

A total of 110 dogs presented to Teaching Veterinary Clinical Complex, CSKHPKV, Palampur (H.P.), India with the history of enteritis/gastroenteritis were screened for Canine Parvovirus (CPV). Out of these 110 dogs, 48 dogs were found positive for CPV by using Scanvet - a rapid antigen detection kit. All the 110 samples were subjected to Polymerase Chain Reaction (PCR) to check whether there were some cases that were false positive or false negative by Scanvet. Positive cases were, then, divided into 4 groups of 12 dogs each, with 4 different treatment regimens followed for each group. In addition to standard treatment protocol, our study tested effect of immunomodulators (Vitamin E & Selenium) and antiviral "Oseltamivir" on recovery of dogs. This type of study (using Oseltamivir and immunomodulators) was not done previously in the state and hence, our aim was to test whether this treatment increases survivability and recovery rate for dogs with Parvoviral enteritis in Himachal Pradesh. Groups containing immunomodulators were more efficacious than their counterparts without immunomodulators. Addition of Oseltamivir and immunomodulators to standard therapy resulted in reduced mortality, cessation of diarrhoea and vomiting earlier than routine therapy. Scanvet diagnostic kit was found to be a quick alternate method for diagnosis of Parvo Viral Enteritis. Overall, the group treated with antibiotic+ supportive+ symptomatic+ antiviral treatment along with immunomodulator displayed promising results.

Keywords: *Dogs, parvo-virus, severe disease, treatment, diagnostic methods, antiviral*

Canine parvovirus enteritis (PVE) is caused due to three mutations in canine parvovirus type 2. These are recognised as CPV2, family Parvoviridae and Genus Parvovirus. During the late 1970s, CPV-2 emerged as the leading cause of global outbreaks of acute canine enteritis (Battilani *et al.,* 2006; Decaro and Buonavoglia, 2012; Greene, 2012; Truyen, 2006). The CPV-2 caused the infections thereafter for almost a decade until it evolved itself into two new variants CPV-2a and CPV-2b (Carmichael, 2005; Martella *et al.,* 2006). Over the next few years, no new mutation was

#Part of the MVSc thesis submitted by the first author to Chaudhary Sarwan Kumar Himachal Pradesh Krishi Vishvavidyalaya (CSKHPKV), Palampur, India

1. *Veterinary officer, Veterinary Hospital-Bah Ki Dhar, Distt. Mandi, Himachal Pradesh, India,*
2. *Assistant Professor, Department of Veterinary Medicine, DGCN, COVAS.*

**Corresponding author: shrutiggupta3@gmail.com, Ph: 09418908800*

recorded worldwide. However, in the year 2000, a third variant CPV-2c was reported in Italy and is being found all around the world ever since except Australia (Buonavoglia *et al*., 2001; Decaro *et al*., 2006; Ntafis *et al*., 2010). All of these variants are believed to contain similar pathogenicity which leads to identical clinical disease (Decaro and Buonavoglia, 2012; Markovich *et al*., 2012). Overall, PVE is a highly contagious and fatal viral disease found in dogs of all ages (Goddard and Leisewitz, 2010; Sykes, 2014).

Although, intense clinical disease commonly occurs in puppies of age less than six months, adults with inadequate immunity may also be potentially affected (Kalli *et al*., 2010; Markovich *et al.,* 2012). The disease manifests in two forms: cardiac and intestinal form. Schatzberg *et al.* (2003) suggested that the cardiac form is only seen in young puppies. Prittie (2004) reported that the intestinal form of disease is characterised by foul smelling, dark bloody faeces, severe vomiting, dehydration, anorexia, depression and can be fatal within 2 days of post infection in very acute cases. Although, a large no. of diagnostic tests viz. Haemagglutination test, ELISA, PCR are available, these are very costly, time consuming (Mosallanejad *et al.,*2008; Oh *et al.,*2006) and need expensive and elaborate laboratory set up. Rapid diagnostic tests offer promise in the cost effective and immediate diagnosis of this disease. However, the authenticity, validity and repeatability of newly developed tests like Scanvet Rapid Parvo Virus Detection kits needs further investigations.

Therapeutic management of parvo viral infection includes the conventional treatment comprising of the use of antibiotics in majority of cases along with supportive and symptomatic therapy (anti-emetics, fluids, electrolytes, intestinal protectants and analgesics). However, the results obtained are highly variable (Goddard and Leisewitz, 2010; Roy *et al.,* 2010). Recently, one antiviral compound "Oseltamivir" originally developed to treat influenza infection in human is also being tried to treat parvo viral infection in dogs. Although several anecdotal reports have come out from practising veterinarians, especially from North America indicating its beneficial effects, only two scientific studies (Savigny and Macintire, 2010; Schatzberg *et al.,* 2003) have been conducted so far to validate the findings. Hence, more systematic studies are required to be undertaken to determine its efficacy. Immunomodulatory approaches using micronutrients, especially, Selenium, Vitamin E and Vitamin C as a non-specific way of maintaining adequate host defense against infection have been mentioned in the treatment of CPV infections and this is in line with Kandil and Zeina (2005).The present communication reports diagnosis and treatment of canine parvo viral infection in dogs in Palampur valley of H.P. using different treatment approaches in order to find appropriate treatment that can ameliorate morbidity and mortality. The main aim of this study was to investigate authenticity and validity of rapid diagnostic tests along with investigating efficiency of new methods of treatment using antiviral and immunomodulators.

Materials and methods

The present study was conducted on a total of 110 dogs presented to Teaching Veterinary Clinical Complex, College of Veterinary and Animal Science, Palampur, with the history of diarrhoea and vomiting. Every dog brought to the clinic was a private pet, and all the owners had given their consent for the treatment. The faecal samples of dogs were screened for parvo viral infection by using Scanvet Canine Parvo virus antigen detection kit developed by Intas Pharmaceuticals Ltd., Ahmedabad (India). The faecal samples were collected from suspected dogs with the help of sterile swab given in the kit by inserting into rectum and followed by the test as per manufacturer's instructions. All the 110 cases were further subjected to PCR in order to detect false positive or false negative by Scanvet kit. For PCR, faecal swabs of all dogs were made bacteria free by filtration with 0.22-micron syringe filter and kept frozen at -20°C. PCR tests were conducted at Department of Veterinary Microbiology, COVAS, Palampur, H.P and further study was done on cases found positive with Scanvet Rapid antigen detection kit.

Polymerase Chain Reaction

Genomic DNA of CPV-2 was extracted by phenol chloroform method from the faecal samples as per the standard method (Sambrook and Russell, 2011). The PCR was standardised for the primer set pCPV 2ab as reported by Pereira *et al.* (2000) with slight modifications (Table 11). The pCPV 2ab primer set amplify portion of VP1/VP2 gene of both CPV 2a and CPV 2b variants (3025 to 3706 nucleotide position of CPV genomic DNA) to yield a product size of 681 bp.

The reaction was optimised using 2.5 µL of 10x Taq DNA polymerase buffer (100mM TrisHCl, pH 8.8, 500mM KCl, 0.8% Nonidet P40, 25mM $MgCl_{2)}$, 200µM of each dNTP, 10 pmol of each forward and reverse primer (0.5µL each), 5µL of processed faecal sample and nuclease free water to make the volume to 25µL.

Therapeutic study

Positive cases as found with Scanvet kit were further included for various therapeutic trial groups. The ailing dogs were placed in 4 different therapeutic groups comprising of 12 animals in each group. The treatment regimens for each group are shown in Table 2.

Symptomatic and supportive therapy consisted of parenteral, anti-emetics, hemostats, B- complex vitamins and fluids i.e., Ringer's lactate and dextrose normal saline. Minimum treatment duration with antimicrobial agent was 3 days in each of the group, extendable up to 5 days, if situation warranted.

All the 48 dogs found positive as per Scanvet kit were first divided into mid-stage and late-stage cases. Cases presented within 2 days of the development of diarrhoea/ dysentery were labelled as mid-stage cases and cases presented after 2 days were labelled as late-stage cases. Out of 48 cases, 16 cases were late-stage and 32 were mid-stage cases. These cases were further divided into 4 treatment regimen groups as per (Table 3).

Statistical analysis

The data obtained were subjected to statistical analysis by using computer software Instat from GraphPad software 2008, California Corporation, and as used by Bhat *et al.* (2013).

Table 3. Group formation of mid-stage and late-stage cases

Group	Mid-stage cases	Late-stage cases
Group I	7	5
Group II	7	5
Group III	9	3
Group IV	9	3

Table 1. Primer sequence used for the amplification of VP1/VP2 gene of CPV

Sr. No.	Forward and Reverse Primers	Primer Sequence	Position in the genome	Annealing temperature and product size
1.	pCPV-2ab (F)	5'-GAA GAG TGG TTG TAA ATA ATT-3' (21 mer)	3025-3045 to 3685-3706	55°C 681bp
2.	pCPV-2ab (R)	5'-CCT ATA TAA CCA AAG TTA GTA C-3' (22 mer)		

Table 2. Treatment regimens

Group	Treatment regimen
Group I	Treated with antibiotics with supportive and symptomatic therapy
Group II	Treated with antibiotics and immunomodulators along with supportive and symptomatic therapy
Group III	Treated with antiviral along with the use of antibiotic, symptomatic and supportive therapy
Group IV	Treated with antiviral and immunomodulator along with using antibiotics, supportive and symptomatic therapy

The mean values of different parameters between control and diseased group; control, pre and post treatment were compared at 1%, 5 % and 10 % level of significance using "t" test and "ANOVA".

Results and discussion

Notwithstanding the fact that 48 cases were found positive both by Scanvet kit and PCR, there were at least 8 such cases which have been found negative by Scanvet and positive by PCR. Figures 1 and 2 below show the results of antigen detection kit, whereas figure 3 shows the results of PCR.

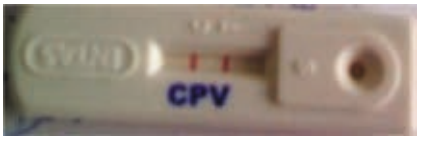

Fig.1. Positive result of rapid antigen detection kit **(**Control and test bands are visible).

Fig. 2. Negative result of rapid antigen detection (Only control band is visible).

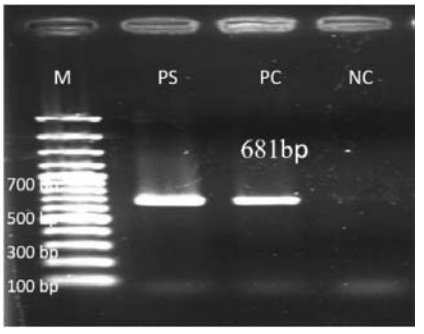

Fig. 3. PCR products: M 100 bp marker, PS positive sample, PC positive control, NC negative control

It means Scanvet kit showed 8 false negative cases (Table 4) that might be due to the requirement of large amount of viral antigen to produce a clearly visible band (Reddy *et al.,* 2015; Tinky *et al.,* 2015) who reported that the quantity of viral particles can affect the immunochromatographic (IC) test result which was observed to be one of the disadvantages of this test.

Therapeutic studies

The consistency, frequency and colour of faeces returned to normal within 3.10 ± 0.51days of initial treatment in dogs of group IV which was the minimum amongst all the four treatment groups. Similarly, the vomiting stopped completely in minimum time of 3.08 ± 0.66 days in dogs of this group. Overall survival rate was also highest viz. 91.67% (11/12) in this group. Number of days taken for cessation of diarrhoea and vomiting was maximum in group I with value of 4.33 ± 0.88 and 4.25 ± 0.96 days respectively. The survivability was also noticed to be minimum i.e., 71.43% (5/7) in dogs of group I. It was worth noting that the survival rate was better in mid-stage cases as compared to late-stage cases in all the treatment groups (Table 5).

All the survived animals regained their normal appetite in 5-7 days of start of treatment and were active, alert and bright in appearance. The total erythrocytic count (TEC) increased significantly ($p< 0.01$) in all the four groups after institution of the treatment. Similarly, the total leucocytic count (TLC) also increased significantly ($p<0.01$) in all the groups. However, the increase was more pronounced in dogs of group III which were given Oseltamivir additionally and in dogs of group IV which were given immunomodulators with Oseltamivir and standard treatment. The pre-treatment mean values of haemoglobin and packed cell volume were recorded to be significantly ($p<0.01$)

Table 4. Comparison of Scanvet kit and PCR test strips

Test		PCR test kit		
	Result	Positive	Negative	Total
Scanvet kit	Positive	48	0	48
	Negative	8	54	62
	Total	56	54	110

lower in all the groups as compared to healthy dogs (Table 6). After institution of treatment, these values were further decreased but the decline was non-significant ($p>0.05$). There was a non-significant ($p>0.05$) increase in the mean values of plasma glucose, thus, showing slight improvement in dogs of all the groups. However, a significant increase ($p<0.01$) was found in blood glucose level of group IV after institution of treatment.

The total plasma protein was found to be lower in dogs of all the four groups even after initiation of the treatment. The blood urea nitrogen (BUN) which was significantly ($p<0.01$) higher in all the parvo virus affected dogs decreased significantly ($p< 0.01$) in all the treatment groups (Table 7). Plasma sodium concentration increased significantly ($p<0.01$) in dogs of all the groups, except group III which showed non-significant increase ($p>0.05$), thus showing improvement in all the four treatment groups. Plasma potassium concentration showed non-significant increase ($p>0.05$) whereas plasma chloride showed significant increase ($p<0.01$) in all four treatment groups indicating gradual improvement.

Number of animals scanned for the CPV is statistically a significant number to conclude that such kits can be a good alternate method for rapid and accurate diagnosis of parvo viral infection especially in field conditions where facilities to perform other tests like PCR and HA are non-existent.

We tried our best to put mid-stage and late-stage cases equally in all 4 treatment groups, so that we can remove any bias in our results. As the number of days taken for cessation of diarrhoea and vomiting were minimum in group IV along with maximum

Table 5. Comparative efficacy and survivability in various treatment protocols for CPV infectious dogs

Group	Days taken for cessation of diarrhoea (Mean ± S.D)	Days taken for cessation of vomiting (Mean ± S.D)	Survivability		
			Mid-stage cases	Late-stage cases	Total survivability
Group I	4.33 ± 0.88	4.25 ± 0.96	71.43% (5/7)	40.00% (2/5)	58.33% (7/12)
Group II	3.66 ± 0.98	3.50 ± 0.67	85.71% (6/7)	60.00% (3/5)	75.00% (9/12)
Group III	3.83 ± 1.02	3.75 ± 0.86	88.89% (8/9)	66.67% (2/3)	83.33% (10/12)
Group IV	3.10 ± 0.51	3.08 ± 0.66	100% (9/9)	66.67% (2/3)	91.67% (11/12)

Table 6. Effect of treatment on haematological parameters (Mean ± S.D.) (n=12) in CPV infectious dogs

Hb (g/dl)	Group I	Group II	Group III	Group IV
Healthy group	12.41 ± 1.37^{b}	12.41 ± 1.37^{b}	12.41 ± 1.37^{b}	12.41 ± 1.37^{b}
Pre-treatment value (0 h)	9.08 ± 1.50^{a}	9 ± 1.47^{a}	9 ± 1.47^{a}	9.08 ± 0.44^{a}
Post-treatment value (72h)	8.25 ± 0.93^{a}	8.41 ± 0.81^{a}	8.50 ± 0.63^{a}	8.75 ± 0.75^{a}
PCV (%)				
Healthy group	36 ± 4.24^{b}	36 ± 4.24^{b}	36 ± 4.24^{b}	36 ± 4.24^{b}
Pre-treatment value (0 h)	27.58 ± 3.44^{a}	26.66 ± 4.61^{a}	26.83 ± 4.62^{a}	26.83 ± 4.62^{a}
Post-treatment value (72 h)	25.75 ± 2.95^{a}	25.58 ± 2.93^{a}	24.75 ± 3.69^{a}	26.16 ± 3.15^{a}
TEC ($\times 10^{6}$/µl)				
Healthy group	5.73 ± 0.87^{b}	5.73 ± 0.87^{b}	5.73 ± 0.87^{b}	5.73 ± 0.87^{b}
Pre-treatment value (0 h)	4.55 ± 0.55^{a}	4.55 ± 0.53^{a}	4.37 ± 0.59^{a}	4.60 ± 0.61^{a}
Post-treatment value (72 h)	5.60 ± 0.74^{b}	5.62 ± 0.74^{b}	5.73 ± 0.75^{b}	5.65 ± 0.64^{b}
TLC ($\times 10^{3}$/ µl)				
Healthy group	9.33 ± 1.65^{b}	9.33 ± 1.65^{b}	9.33 ± 1.65^{b}	9.33 ± 1.65^{b}
Pre-treatment value (0 h)	5.60 ± 0.74^{a}	5.50 ± 1.09^{a}	4.81 ± 0.42^{a}	5.01 ± 0.45^{a}
Post-treatment value (72 h)	11.48 ± 0.66^{c}	11.79 ± 1.28^{b}	12.78 ± 0.66^{c}	13.08 ± 0.48^{c}

Values with different superscripts in a column differ significantly ($p<0.01$)

Table 7. Effect of treatment on blood biochemical profile (Mean ± S.D.) (n=12) in CPV infectious dogs

Glucose (mg/dl)	Group I	Group II	Group III	Group IV
Healthy group	105.58 ± 6.08^{b}	105.58 ± 6.08^{b}	105.58 ± 6.08^{b}	105.58 ± 6.08^{b}
Pre-treatment value (0 h)	81.08 ± 7.36^{a}	81.83 ± 6.87^{a}	78.41 ± 7.31^{a}	79.41 ± 6.31^{a}
Post-treatment value (72 h)	84.08 ± 6.82^{a}	87.08 ± 4.27^{a}	79.25 ± 6.55^{a}	86.75 ± 6.57^{c}
Total Protein (g/dl)				
Healthy group	6.60 ± 0.59^{b}	6.60 ± 0.59^{b}	6.60 ± 0.59^{b}	6.60 ± 0.59^{b}
Pre-treatment value (0 h)	4.86 ± 0.95^{a}	4.95 ± 0.88^{a}	4.78 ± 0.78^{a}	4.61 ± 0.67^{a}
Post-treatment value (72 h)	4.60 ± 0.66^{a}	4.79 ± 0.77^{a}	4.45 ± 0.47^{a}	4.40 ± 0.42^{a}
BUN (mg/dl)				
Healthy group	12.0 ± 2.0^{a}	12.0 ± 2.0^{a}	12.0 ± 2.0^{a}	12.0 ± 2.0^{a}
Pre-treatment value (0 h)	25.91 ± 3.26^{b}	26.75 ± 4.80^{b}	25.08 ± 3.39b	26.58 ± 4.79^{b}
Post-treatment value (72 h)	13.25 ± 1.65^{a}	12.83 ± 1.69^{a}	13.41 ± 1.50^{a}	11.83 ± 1.40^{a}
Sodium (mmol/l)				
Healthy group	147.33 ± 4.63^{b}	147.33 ± 4.63^{b}	147.33 ± 4.63^{b}	147.33 ± 4.63^{b}
Pre-treatment value (0 h)	126.33 ± 4.37^{a}	126.33 ± 4.37^{a}	133.58 ± 5.74^{a}	128.66 ± 5.21^{a}
Post-treatment value (72 h)	136.33 ± 4.09^{c}	136.41 ± 4.12^{c}	139.66 ± 4.51^{a}	140.83 ± 3.73^{c}
Potassium (mmol/l)				
Healthy group	4.15 ± 0.45^{a}	4.15 ± 0.45^{a}	4.15 ± 0.45^{a}	4.15 ± 0.45^{a}
Pre-treatment value (0 h)	3.50 ± 0.29^{b}	3.55 ± 0.28^{b}	3.59 ± 0.25^{b}	3.61 ± 0.15^{a}
Post-treatment value (72 h)	3.73 ± 0.15^{b}	3.76 ± 0.14^{b}	3.84 ± 0.10^{b}	3.89 ± 0.80^{a}
Chloride (mmol/l)				
Healthy group	113.50 ± 2.93^{b}	113.50 ± 2.93^{b}	113.50 ± 2.93^{b}	113.50 ± 2.93^{b}
Pre-treatment value (0 h)	93.33 ± 2.99^{a}	93.25 ± 2.89^{a}	93.33 ± 2.99^{a}	94.16 ± 2.72^{a}
Post-treatment value (72 h)	98.93 ± 2.79^{c}	98.75 ± 2.59^{c}	98.75 ± 2.59^{c}	98.25 ± 2.14^{c}

Values with different superscripts in a column differ significantly ($p<0.05$)

survivability, the treatment regimen of this group gave best results in our study. However, treatment regimen of group I showed least survivability among all four treatment groups and took maximum days for cessation of diarrhoea and vomiting. As the survival rate was better in mid-stage cases as compared to late-stage cases in all the treatment groups, thus, it can be concluded that chances of recovery are lesser in advanced cases.

A variety of treatment protocols have been suggested and adopted by various researchers to treat the parvo virus affected dogs (Chakrabarti, 2012; de Mari *et al.,* 2003; Roy *et al.,* 2010). However, wide variation has been noticed with regard to their efficacy. Many clinicians have advocated extensive use of symptomatic and supportive therapy, especially fluids for successful treatment of parvo virus infection as reported by Prittie (2004); however, Roy *et al.* (2010) have recommended the additional use of antibiotics (Ceftriaxone and Tazobactam) along with supportive therapy to obtain the better response. Navya *et al.* (2022) suggested supplementation of intravenous sodium bicarbonate in treatment of parvoviral enteritis. Savigny and Macintire (2010) carried out detailed investigation of the use of Oseltamivir to treat the parvo virus affected dogs. A significant weight loss during hospitalization as well as significant decrease in WBC count were noticed in control group as compared to treatment group and no major adverse effects were identified that could be associated with Oseltamivir administration (Savigny and Macintire, 2010). Our study showed higher survivability rates in Oseltamivir treated groups which has been shown in table 6.

Oseltamivir is an antiviral drug, basically, developed for the treatment of human influenza. It acts on the influenza (RNA) virus by way of inhibition of neuraminidase enzyme, thus preventing the virus replication in the body as reported by Gubareva *et al.* (2000). Parvo virus is a DNA virus and it does not rely upon

neuraminidase for its replication/pathogenicity and thus, Oseltamivir does not possess direct antiviral activity on this virus. There are some scientific studies especially one in North America that has shown beneficiary effect of the drug in parvo virus affected dogs reported by Savigny and Macintire (2010).This may be due to some indirect actions including inhibition of adherence, translocation and colonisation of secondary bacterial infections and thus, preventing the sepsis and endotoxaemia and systemic inflammatory response and eventual organ failure that is thought to be the main mechanism behind the mortality of CPV enteritis (Crawford and Sellon, 2010; Macintire and Douglass, 2006).

In the light of above therapeutic observations, it was inferred that addition of Oseltamivir (anti-viral drug) and immunomodulators (Selenium and Vitamin E) in the already prevailing conventional therapy (antibiotics + symptomatic and supportive therapy) resulted in higher recovery and survivability in parvo virus affected dogs. Total leucocytic count increased significantly in the groups treated with Oseltamivir as well as Oseltamivir + immunomodulators. Thus, it can be inferred that addition of Oseltamivir as anti-viral, and Vitamin E and Selenium as immunomodulators provide better results as far as survivability and cessation of symptoms (diarrhea and vomiting) is concerned. It is further added that we have not applied any statistical test on survivability, and hence further studies are warranted to validate the above findings covering more animals and a wider geographical area.

Conclusion

Scanvet rapid antigen detection kit is a quick alternate method for diagnosis of parvo viral enteritis in field conditions where PCR can't be used. Above findings suggest that addition of Oseltamivir (anti-viral drug) and immunomodulators (Selenium and Vitamin E) in the already prevailing conventional therapy (antibiotics + symptomatic and supportive therapy) delivers higher recovery and survivability in parvo virus affected dogs. Thus, it can be concluded that addition of Oseltamivir and immunomodulars provide better results as far as survivability and cessation of symptoms is concerned.

References

Battilani, M., Bassani, M., Forti, D. and Morganti, L. 2006. Analysis of evolution of Feline Parvo Virus. *Vet. Res. Commun.* **30**: 223–226.

Bhat, A.A., Wadhwa, D.R., Singh, S.P. and Singh, I. 2013. Haematological and biochemical analysis in canine enteritis. *Vet World, **6**:380*-383.

Buonavoglia, C., Martella, V., Pratelli, A., Tempesta, M., Cavalli, A., Buonavoglia, D., Bozzo, G., Elia, G., Decaro, N. and Carmichael, L. 2001. Evidence for evolution of canine parvovirus type 2 in Italy. *J. Gen. Virol.* **82**(12): 3021–3025.

Carmichael, L.E.2005. An annotated historical account of canine parvovirus. *J. Vet. Med. B*. **52**(7-8):303–311.

Chakrabarti, A. 2012. Canine Parvo Viral Enteritis. In: *Textbook of Clinical Veterinary Medicine,* Oscar publication, Delhi, India.

Crawford, C. and Sellon, R.K. 2010. Canine viral diseases. In: *Textbook of Veterinary Internal Medicine-Diseases of dog and cat* (7thEd.), Vol-I, Chapter 216. Ettinger, S.J. and Feldman, E.C.

Decaro, N., Martella, V., Desario, C., Bellacicco, A.L., Camero, M., Manna, L., d'Aloja, D. and Buonavoglia, C. 2006. First detection of canine parvovirus type 2c in pups with haemorrhagic enteritis in Spain. *J. Vet. Med. B.* **53**(10): 468–472.

Decaro, N. and Buonavoglia, C. 2012. Canine parvovirus: A review of epidemiological and diagnostic aspects, with emphasis on type 2c. *Vet. Microbiol*. **155**(1):1–12.

de Mari, K., Maynard, L., Eun, H.M. and Lebreux, B. 2003. Treatment of canine parvoviral enteritis with interferon-omega in a placebo-controlled field trial. *Vet Rec.* **152**:105-108.

Goddard, A. and Leisewitz, A.L. 2010. Canine Parvovirus. *Vet. Clin. North Am. Small Anim. Pract.* **40**:1041-1053.

Greene, C.E. 2012. Feline enteric viral infections. In: *Greene CE, editor*. *Infectious Diseases of the Dog and Cat*. (4thEd.) St Louis, MO-Elsevier Saunders: 80–91.

Gubareva, L.V., Kaiser, L. and & Hayden, F.G. 2000. Influenza virus neuraminidase inhibitors. *Lancet.* **335**:827-835.

Kalli, I., Leontides, L.S., Mylonakis, M.E., Adamama-Moraitou, K., Rallis, T. and Koutinas, A.F. 2010. Factors affecting the occurrence, duration of hospitalization and final outcome in canine parvovirus infection. *Res. Vet. Sci.* **89**(2): 174–178.DOI: 10.1016/j. rvsc.2010.02.013.

Kandil, O.M and Zeina, H.A. 2005. Effect of Parenteral Vitamin E and Se supplementation on immune status of dogs vaccinated with subunit and somatic antigens against *Taenia hydatigena*. *J. Egypt Soc. Parasitol.* **35**(2):537-50.

Macintire, D.K. and Douglass, K. 2006. Treatment of parvo viral enteritis. In: *Proceedings of CVC Veterinary Conference,* Kansas City.

Markovich, J.E., Stucker, K.M., Carr, A.H., Harbison, C.E., Scarlett, J.M. and Parrish, C.R. 2012. Effects of canine parvovirus strain variations on diagnostic test results and clinical management of enteritis in dogs. *J. Am. Vet. Med. Assoc.* **241**(1):66–72. DOI: 10.2460/ javma.241.1.66.

Martella, V., Decaro, N. and Buonavoglia, C. 2006. Evolution of CPV-2 and implication for antigenic/genetic characterization. *Virus Genes*. **33**(1):11–13.

Mosallanejad, B., Ghorbanpoor, N.M., Avizeh, R. and Ronagh, A. 2008. Prevalence of Canine Parvo Virus infection in diarrheic dogs referred to veterinary hospital in Ahvaz. *Arch. Razi Inst.* **63**(2):41-46.

Navya, E., Vinodkumar, K., Justin Davis, K., Priya P.M., Ashmy, K. and Vijayakumar K. 2022. Prognostication of haemato-biochemical, electrolyte and blood gas parameters in canine parvoviral enteritis. *J. Vet. Anim. Sci.* **53**(3): 333-339. https:// doi.org/10.51966/jvas.2022.53.3.333-339.

Ntafis, V., Xylouri, E., Kalli, I., Desario, C., Mari, V., Decaro, N. and Buonavoglia, C. 2010. Characterization of canine parvovirus type 2 (CPV-2) variants circulating in Greece. *J. Vet. Diagn. Invest.* **22**(5): 737–740. DOI-10.1177/104063871002200512.

Oh, J.S., Ha. G.W., Cho, Y.S., Kim, M.J., An, D.J., Hwang, K.K., Lim, Y.K., Park, B.K., Kang, B. and Song, D.S.2006. One-step immunochromatography assay kit for detecting antibodies to canine parvo virus. *Clin. Vaccine Immunol.* **13**(4): 520-524.

Pereira, C.A., Monezi, T.A., Mehnert, D.U., Angelo,M. D'. and Durigon, E. L. 2000. Molecular characterization of Canine Parvo Virus in Brazil by PCR. *Vet. Microbiol.* **75**(2):127-133.

Prittie, J. 2004. Canine parvo viral enteritis: A review of diagnosis, management and prevention. *J. Vet. EmergCrit Care* **14**:167-176.

Reddy K, Basava., Shobhamani, B., Sreedevi, B. and Prameela, D. Rani. 2015. Diagnosis of Canine Parvo Viral (CPV) Infection in Dogs. *IntasPolivet* **16**(2):441-442.

Roy, S., Roy, M. and Sagar, K.A. 2010. Haemato-biochemical and therapeutic studies of canine parvo viral infection. *IntasPolivet* **11:**344-347.

Sambrook, J. and Russell, D. 2011. *Molecular Cloning: A Laboratory Manual* (3rdEd.), Cold Spring Harbour Laboratory Press-USA.

Savigny, M.R. and Macintire, D.K.2010 Use of Oseltamivir in the treatment of canine parvoviral enteritis. *J. Vet. EmergCrit Care* **20**:132-142.

Schatzberg, S.J., Haley, N.J., Barr, S.C., Parrish, C., Steingold, S., Summers, B.A., deLahunta, A., Kornegay, J.N. and Sharp, N.J.H. 2003. Polymerase Chain Reaction Amplification of Parvoviral DNA from the brains of dogs and cats. *J. Vet. Intern. Med.***17**(4): 538-544.DOI: 10.1111/j.1939-1676.2003. tb02475.x.

Sykes, J.E. 2013. Canine parvovirus infections and other viral enteritides. In: *Sykes JE, editor-Canine and Feline Infectious Diseases*, (1stEd.), St Louis, MO: Elsevier: 141-151.

Tinky, S.S., Ambily, R., Nair, S.R. and Mini, M. 2015. Utility of a rapid immunochromatographic strip test in detecting canine parvovirus infection compared with polymerase chain reaction. *Vet. World* **8**(4):523-526.

Truyen, U. 2006. Evolution of canine parvovirus-a need for new vaccines? Vet. Microbiol. 117(1):9-13. ■