



Canine Parvovirus Infection in Dogs: Prevalence and Associated Risk Factors in Egypt

Mohamed Z. Sayed-Ahmed^{1,2*}, Elzahara Elbaz¹, Emad Younis¹, and Mohamed Khodier³

¹Department of Internal Medicine and Infectious Diseases, Faculty of Veterinary Medicine, Mansoura University, Mansoura 35516, Egypt

²Department of Clinical Pharmacy, College of Pharmacy, Jazan University, Jizan 45142, Saudi Arabia

³Pet Animal Vaccine Department, Veterinary Serum and Vaccine Research Institute, Abbasia 11384, Cairo, Egypt

* Corresponding author's E-mail: drzakaria-infect@hotmail.com; ORCID: 0000-0003-0777-8256

ABSTRACT

Canine parvovirus (CPV) infection is a global infectious and contagious viral disease of canine, especially in dogs infected by three variants of CPV type. This study aimed to investigate the prevalence and potential risk factors of parvovirus infection in dogs residing in Egypt. A total of 122 dogs suffering from vomiting and diarrhea were screened by antigen rapid CPV/Canine Coronavirus Ag test kit for the diagnosis of CPV infection from March 2012 to February 2013. Age, breed, season, and vaccination of each dog were recorded to study the prevalence of CPV. The overall prevalence of CPV infection in dogs was reported as 59.7%. Dogs between 0 and 3 months of age indicated the highest prevalence of 68% followed by 4-6 months of age which was 53.3%. The lowest prevalence of CPV was reported in dogs above 6 months of age (20%). The maximum prevalence was noticed in non-descript dogs (48.5%) followed by German shepherds (26.7%), Doberman (23.07%), and Griffon (16.6%). Among different risk factors, young, unvaccinated puppies and exotic breeds were more prone to CPV infection. Regarding the season, the higher prevalence was noticed in summer (77.1%) followed by spring (55.5%), autumn (25%), and winter (16.6%). Thus, CPV is an infectious and highly contagious viral disease of dogs. Age and seasonal variations are risk factors in the prevalence of CPV infection. Identification of the potential risk factors associated with the disease may be helpful to construct the ideal preventive measures.

Keywords: Canine parvovirus, Egypt, Epidemiology, Prevalence, Risk factors

INTRODUCTION

Canine parvovirus (CPV) infection is a global infectious and contagious viral disease of canine species especially dogs caused by three variants of canine parvovirus type 2 (Goddard and Leisewitz, 2010; Sykes, 2014; Khare et al., 2019). Canine parvovirus-2 (CPV-2) is the cause of acute enteritis associated with high morbidity and mortality, with very low survival rates in untreated dogs. Although the severity of disease typically occurs in dogs younger than 6 months of age, the immunosuppressed adults may potentially be affected (Decaro et al., 2004; Marcovich et al., 2012; Albaz et al., 2015; Mylonakis et al., 2016; AL-hosary, 2016; Khare et al., 2019). Seasonal prevalence of the disease is affected by geographic variation (Kalli et al., 2010). CPV-2 can survive more than one year in the environment and infect the susceptible dogs through infected feces, or vomitus by fecal-oral route (Decaro et al., 2005a; Sykes, 2014; Albaz et al., 2015). The incubation period following natural or experimental exposure ranges from four to fourteen days, and virus shedding starts a few days before the occurrence of clinical signs, progressively declining 3 to 4 weeks post-exposure (Decaro et al., 2005b; McCaw and Hoskins, 2006). The clinical signs of CPV infection depend on the age and immune status of the dogs, dose of the virus, virulence of the virus, and pre-existing infections (McAdaragh et al., 1982). CPV-2 infection manifests as acute hemorrhagic enteritis and myocarditis. Dogs with enteritis show high fever, depression, loss of appetite, lethargy, vomiting, and severe mucoid or bloody diarrhea (Prittie, 2004; Lamm and Rezabek, 2008; Albaz et al., 2015; Khare et al., 2019). Factors that predispose puppies to parvovirus infection are lack of maternal immunity, gastrointestinal parasites, unsanitary, and stress (Hong et al., 2007; Mylonakis et al., 2016). A definitive diagnosis of CPV-2 in the feces of affected dogs is done by viral isolation, fecal hemagglutination, latex agglutination, immunochromatography, PCR, and electron microscopy (Pollock and Carmichael, 1988; Desario et al., 2005; Wilkes et al., 2015). CPV infection can be treated by symptomatic and supportive therapy (Prittie, 2004; Brown and Otto, 2008; Mylonakis et al., 2016) involving antiemetic, antibiotics, nutritional support, fluid therapy, antiviral treatments, and pain management. Prevention is by vaccination of dogs with either attenuated or modified live vaccines (Martella et al., 2005; Albaz et al., 2015; Mylonakis et al., 2016). Hence, the present study was aimed to know the prevalence and potential risk factors of parvovirus infection in dogs in Egypt.

ORIGINAL ARTICLE
 pii: S232245682000068-10
 Received: 29 Oct 2020
 Accepted: 13 Dec 2020

MATERIALS AND METHODS

Ethical approval

This study was certified, approved and performed according to the ethics of committee of the Faculty of veterinary medicine, Mansoura University, Mansoura, Egypt.

Study area and animals

For one year, an epidemiologic study was carried out at veterinary teaching hospital, faculty of veterinary medicine, Mansoura University, Egypt from March 2012 to February 2013. One hundred twenty-two dogs of different breeds within the age ranging from one day up to 12 months which distributed all over different localities were subjected for clinical examination where the noticed signs were suspected to be parvovirus infection including fever, diarrhea and vomiting, tachycardia, dehydration, and weakness.

Case definition

The diagnosis was made by anamnesis and clinical signs observed. In CPV infection, the main clinical signs were high fever (104-105°F), vomiting, and bloody diarrhea. If a dog showed signs of high fever, vomiting, bloody diarrhea, anemia, dehydration, it would be suspected as a CPV infection. Anemia was detected by the pale mucous membrane. The degree of dehydration was estimated by a skin fold test (Hasan et al., 2018).

Clinical examination and data collection

The questionnaire was developed according to age, sex, breed, history of vaccination, clinical history, and the data were collected by interviewing the owner. Rectal temperature, heart rate, and respiration rate of the sample dogs were measured. Skinfold test was performed to estimate the degree of dehydration. Then the clinical signs and symptoms were observed. All the clinical signs and symptoms were separately recorded for each clinical case (Kelly, 1990; Hasan et al., 2018).

Samples and sample processing

Fecal samples

Fecal swabs were collected from diseased puppies; each swab was inserted into a screw-capped bottle containing 3 ml of sterilized phosphate buffer saline containing 100 IU of penicillin as well as 100 mg of streptomycin/ml. The swabs were squeezed and removed. All samples were centrifuged at 2000 rpm for 10 minutes. The supernatant solution was transferred to a sterile bijoux bottle and the samples were preserved at -20°C until being examined (Coles, 1986; Wilkes et al., 2015; AL-hosary, 2016).

Blood sample

Two blood samples were collected from diseased puppies via the femoral vein, one with anticoagulant virus isolation and the other without anticoagulant for serological examination.

Viral isolation and identification

Vero cells (African green monkey kidney cells) were used in the virus isolation procedures. Dr. J. House, Plum Island, USA, kindly supplied it. The samples were inoculated at a rate of 100 µL per well in a cell's monolayer in the maximum growth phase (12 hours of culture) with a low cell confluence (60%). After being adsorbed for 1 hour at 37°C, the inoculum was removed, and the minimum essential medium with Eagle salts was added (E-MEM, Sigma-Aldrich®, USA), then the cells were again incubated at 37°C. Cytopathic effect was monitored daily in the cell culture for 4-5 days. Subsequently, the plate was frozen at -80°C, and submitted to further passages following the same procedure, until the fifth passage or the eventual appearance of cytopathic effect. The cells were examined for the presence of virus by the immunofluorescence according to a 2-day protocol with minor variations according to *Virology Manual, 2014*, Immunochromatography assay for the qualitative detection of CPV antigen in fecal or rectal swab as described by Schmitz et al. 2009, and Faz et al. 2017 and virus neutralization test according to Rossitter and Jessett (1982), and Hao et al. 2017.

Statistical analysis

All the data including age, breed, sex, bloody diarrhea, vaccination, dehydration, and diagnosis were entered into Microsoft Office Excel-2010. Then the data was cleaned, coded, recoded, and finally analyzed using statistical software STATA Version-11 (STATA Corporation, College Station, Texas). Prevalence was calculated according to different categories of the explanatory variables. A Chi-square (χ^2) test was performed to identify the association between a categorical variable with the occurrence of CPV infection. The association was regarded as significant if the p value was ≤ 0.05 , and highly significant when p value was 0.01.

Table 1. Analysis of diagnosed canine parvovirus cases based on age, breed, season and vaccination status, presented to the veterinary teaching hospital, faculty of veterinary medicine, Mansoura University, Egypt from 2012 to 2013

Variable	Category	No of examined animals; n: 67	Positive case	Proportionate prevalence	P value
Age	1-3 month	47	31	65.9	0.068
	4-6 month	15	8	53.3	
	>6 month	5	1	20	
Breed	non-descript dogs	33	16	48.4	0.589
	German shepherd	15	4	26.7	
	Doberman	13	3	23.07	
	Griffon	6	1	16.6	
Season	Summer	35	27	77.1	0.222
	Spring	18	10	55.5	
	Autumn	8	2	25	
	Winter	6	1	16.6	
Vaccination	Vaccinated	26	5	19.2	0.163
	Unvaccinated	41	24	58.5	

RESULTS

Among the 67 clinically sick dogs, 40 were found positive for CPV infection. Prevalence of different risk factors (age, breed, season, localities) associated with CPV disease is summarized in (Table-1). The study showed that the overall prevalence of CPV infection was 59.7%. The prevalence of CPV infection in different age groups differed insignificantly ($P \leq 0.05$), and these were 65.9% for the age range of 1 to 3 months, 53.3% for 4 to 6 months, and 20% for above 6 months of ages. In the table 2, the maximum prevalence was noticed in non-descript dogs (48.5%) followed by German shepherds (26.7%), Doberman (23.07%), and Griffon (16.6%). Among different risk factors young, unvaccinated puppies and exotic breeds were more prone to CPV infection. Regarding the season, the higher prevalence was noticed in summer (77.1%) followed by spring (55.5%), autumn (25%), and winter (16.6%). Among the breeds, the rate of infections was encountered as 48.4% in native dogs, 26.7% in German shepherds, 23.07% in Dobermans, and Griffon (16.6%) were differed insignificantly ($P \leq 0.05$). The study revealed that in CPV infected dogs, 68.6% with diarrhoea, 17.2% with vomiting, 10.7% with tachycardia, and 3.3% with sudden death were recorded (Figure-1). Moreover, 19.2% of vaccinated dogs and 58.9% of non-vaccinated dogs were significantly ($P \leq 0.05$) affected with CPV infection.

Table 2. Different clinical signs observed among the canine parvovirus cases presented to the veterinary teaching hospital, faculty of veterinary medicine, Mansoura university, Egypt from 2012 to 2013); n= 122

Variable	Category	Positive case	Percentage of positive cases (%)
Bloody diarrhea	Yes	30	24.5
	No	92	75.5
Mucoïd diarrhea	Yes	54	44.2
	No	68	55.8
Vomiting	Yes	21	17.2
	No	101	82.8
Tachycardia	Yes	13	10.7
	No	109	89.3
Sudden death	Yes	4	3.3
	NO	118	96.7

n= number of examined animals

DISCUSSION

The overall prevalence of CPV infection was found to be 59.7%. Archana et al. (2010), Roy et al. (2010), and Khare et al. 2019 reported similar higher prevalence 45.30% and 65.04% in Jabalpur and Chhattisgarh, respectively. In contrast to the present findings, Wazir et al. (2013) reported a lower prevalence in Jammu who found a 6.93% prevalence of CPV infection. The present findings indicated the presence of CPV in dogs. An immuno-chromatographic assay-based kit was useful in supporting the diagnosis, and it was also useful particularly in epidemiological studies. The variation in the prevalence of CPV might be due to the diagnostic tests variation among different studies. Due to the wide variation in

the number of samples tested by the different workers in the different geographically areas, comparison in this regard would be of little value. However, these variations observed in the prevalence were difficult to explain due to the different study areas and differences in the methods of sample analysis. The age prevalence of CPV infection revealed that maximum prevalence in the dogs of 0 to 3 months of age was 65.9%, followed by 4 to 6 months of age was about 53.3%, and > 6 months of age was 20%. These finding agree with Sakulwira et al. (2003) and Al-hosary (2016) that reported a higher prevalence of CPV infection in 3 to 6 months old dogs. However, Roy et al. (2010), Mukhopadhyay et al. (2012), Dongre et al. (2013) and Khare et al. (2019) who stated that, the age wise prevalence of CPV infection revealed maximum prevalence in the dogs of 0-3 months of age i.e. 11.9%, followed by 3-6 months of age i.e. 7.09%, 6-12 months of age i.e. 5.31% and above 12 months of age i.e. 1.11%. The higher prevalence in the dogs of 0 to 3 months of age may be attributed to the higher susceptibility of enterocytes to the viral tropism. Houston et al. (1996) stated that during weaning, enterocytes of the intestinal crypts have a higher mitotic index because of the changes in bacterial flora and diet, and were therefore more susceptible. Thus, the higher prevalence of CPV infection in young dogs (0 to 3 months) was probably because of the close affinity of the virus with rapidly dividing cells of the intestine, which decline with the advancement of age (Banja et al., 2002; Khare et al., 2019). Above one year age, very few incidences was recorded which might be possible due to developing antibodies in the adults either due to vaccination schedule practiced or due to mild exposure to virus leading to build up antibody in the host or some other reasons that need to be explored. Canine Parvovirus infection was reported in the various breeds of the dogs including German shepherds, Doberman, Griffon, and non-descript dogs. The maximum prevalence was noticed in non-descript dogs which was about 48.4% followed by a German shepherd, Doberman, and Griffon in which prevalence was found to be 26.7%, 23.07% and 16.6% respectively. The higher prevalence in non-descript dogs was also reported by Tajpara (2003), Archana et al. (2010) and Wazir et al. (2013), while, Roy et al. (2010), Dongre et al. (2013), Al-hosary (2016) and Khare et al. (2019) reported a higher prevalence in German shepherds breed of dogs. The higher prevalence in non-descript breeds might be due to the higher population density of this breed making their proximity to spread the infection or poor vaccination schedule being followed by the owners of the non-descript breeds due to the lack of awareness among them. No specific comment can be made on breed susceptibility as the population density of the breed varies from one geographical area to another (Archana et al., 2010; Khare et al., 2019). The prevalence was higher in non-vaccinated dogs compared to the vaccinated ones. The finding was in agreement with (Godsall et al., 2010) where unvaccinated puppies aged between six weeks and six months were at greatest risk of developing CPV infection. The higher prevalence of CPV infection in non-vaccinated dogs might be due to a lack of protective immunity. In vaccinated dogs, CPV infection might occur due to incomplete or ineffective primary vaccination course, or a failure of vaccination. The main clinical signs of CPV disease are bloody diarrhea, vomiting, and dehydration. The findings of present study were in agreement with Thomson and Gagnon (2005), Prittie (2004) and Khare et al. (2019). Bloody diarrhea and vomiting were observed in 68.7% and 17.2% of CPV positive dogs respectively. A similar finding was also reported previously by Thomson and Gagnon (2005), Mylonakis et al. (2016). 84.28% of CPV positive dogs had severe dehydration which was supported by the previous study (Laforcade et al., 2003; Mylonakis et al., 2016).



Figure 1. Different clinical signs observed among the dogs infected with canine parvovirus virus. Vomiting (A), bloody diarrhea (B), mucoïd diarrhea (C), and hemorrhagic enteritis (D).

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. Both non-descript dogs and the breeds like German shepherds, Dobermans and Griffon are susceptible to CPV infection. The rate of the infection was higher in non-vaccinated than vaccinated dogs. So, the identification of the potential risk factors of the disease may be helpful to construct the ideal preventive measures.

DECLARATIONS

Acknowledgments

The authors express their sincere gratitude to Pet Animal Vaccine Department, Vaccine and Serum Research Institute, Abbasia, Cairo, Egypt for all the support provided in laboratory experiments. Funding of this study was on the expense of the authors and did not receive any funds from any agencies. Nonetheless, the authors acknowledged the support and cooperation of the administration and staff of the Department of Internal Medicine and Infectious Diseases, Faculty of veterinary Medicine, Mansoura University, Egypt.

Competing interests

All authors declare no competing interests that might interfere with the data provided in the current manuscript.

Author's contributions

Manuscript preparation, writing-reviewing, editing and statistical analysis were conducted by Mohamed Sayed-Ahmed. All authors have contributed to lab work, and the experimental design. All authors read, revised and approved the final manuscript.

Consent to publish

All the authors approved and agreed to publish the manuscript

REFERENCES

- Albaz A, Sayed-Ahmed M, Younis E and Khodier M (2015). Investigation of the antiviral effect of acyclovir on canine parvovirus infection. *Pharmacy Pharmacology International Journal*, 2(2): 14. DOI: <http://dx.doi.org/10.15406/ppij.2015.02.00014>
- Al-hosary AAT (2016). Prevalence of Parvovirus Infection in Household Dogs with Special Reference to its Effects on Some Blood Parameters. *Alexandria Journal of Veterinary Sciences*, 51(2): 174-177. DOI: <http://dx.doi.org/10.5455/ajvs.236999>
- Archana S, Gupta DK and Kumar B (2010). Epidemiology of canine parvovirus infection. *Indian Journal of Veterinary Research*, 18: 42-44. DOI: <http://dx.doi.org/10.14202/vetworld.2015.33-37>
- Banja BK, Sahoo N, Das PK and Ray SK (2002). Clinico-therapeutic aspects of gastroenteritis in dogs. *Indian Veterinary Journal*, 79: 837-840. Available at: <https://agris.fao.org/agris-search/search.do?recordID=IN2004000441>
- Brown AJ and Otto CM (2008). Fluid therapy in vomiting and diarrhea. *Veterinary Clinics of North America: Small Animal Practice*, 38(3): 653-75. DOI: <http://dx.doi.org/10.1016/j.cvs.2008.01.008>
- Burleson FG, Chambers TM; Wiedbrauk DL (2014). *Virology: a laboratory manual*. 3rd ed. Saint Louis: Elsevier Science pp. 223-233. Available at: <https://www.worldcat.org/title/virology-a-laboratory-manual/oclc/1041746460?referer=di&ht=edition>
- Coles EH (1986). *Veterinary clinical pathology* 4th edition. W. B. Saunders Company, Philadelphia, London and Toronto, pp. 1-8. Available at: https://openlibrary.org/books/OL2531334M/Veterinary_clinical_pathology
- Decaro N, Desario C, Campolo C, Cavalli A, Ricci D, Martella V, Tempesta M and Buonavoglia C (2004). Evaluation of the lactogenic immunity to canine parvovirus in pups. *New Microbiologica*, 27: 375-379. Available at: <https://pubmed.ncbi.nlm.nih.gov/15646052/>
- Decaro N, Elia G, Martella V, Desario C and Campolo M (2005a). A real-time PCR assay for rapid detection and quantitation of canine parvovirus type 2 DNA in the feces of dogs. *Veterinary Microbiology*, 105: 19-28. DOI: <http://dx.doi.org/10.1016/j.vetmic.2004.09.018>
- Decaro N, Desario C, Campolo M, Elia G, Martella V, Ricci D, Lorusso E and Buonavoglia C (2005b). Clinical and virological findings in pups naturally infected by canine parvovirus type 2 Glu-426 mutant. *Journal of Veterinary Diagnostic Investigation*, 17(2): 133-138. DOI: <http://dx.doi.org/10.1177/104063870501700206>

- Desario C, Decaro N, Campolo M, Cavalli A, Cirone F, Elia G, Martella V, Lorusso E, Camero M and Buonavoglia C (2005). Canine parvovirus infection: which diagnostic test for virus. *Journal of Virological Methods*, 126(1): 179-185. DOI: <http://dx.doi.org/10.1016/j.jviromet.2005.02.006>
- Dongre J, Mehta H and Maheshwari P (2013). Hematological changes in dogs affected with canine parvo virus Infection, *Veterinary Practitioner*, 14(2): 235-238. Available at: <http://www.vetpract.in/Archives/Dec-2013/VP-14-2--DEC-2013/>
- Faz M, Martínez JS, Quijano-hernández I and Fajardo R (2017). Reliability of clinical diagnosis and laboratory testing techniques currently used for identification of canine parvovirus enteritis in clinical settings. *The Journal of Veterinary Medical Science*, 79(1): 213–217. DOI: <http://dx.doi.org/10.1292/jvms.16-0227>
- Goddard A and Leisewitz AL (2010). Canine parvovirus. *Veterinary Clinics of North America: Small Animal Practice*, 40(6): 1041-1053. DOI: <http://dx.doi.org/10.1016/j.cvsm.2010.07.007>
- Godsall S, Clegg S, Stavisky J, Radford A and Pinchbeck G (2010). Epidemiology of canine parvovirus and coronavirus in dogs presented with severe diarrhoea to PDSA Pet Aid hospitals, *Veterinary Record*, 167(6): 196-201. DOI: <http://dx.doi.org/10.1136/vr.c3095>
- Hao L, Wang Y, Bai C, Jin Y, Zheng D, Chen L, Li X, Deng J and Tian K (2017). Preparation and Application of two Monoclonal Antibodies against Canine Parvovirus Vaccine and Field Strains. *Journal of Vaccines and Immunology*, 3(1): 1-4. DOI: <https://doi.org/10.17352/jvi.000020>
- Hong C, Decaro N, Desario C, Tanner P, Camila Pardo M, Sanchez S, Buonavoglia C and Saliki JT (2007). Occurrence of canine parvovirus type 2c in the United States *Journal of Veterinary Diagnostic Investigation*, 19(5): 35-539. DOI: <http://dx.10.1177/104063870701900512>
- Houston DM, Ribble CS, and Laurie L (1996). Risk factors associated with Parvovirus enteritis in dogs: 283 cases. *Journal of American Veterinary Medicine Association*, 208: 542-546. Available at: <https://pubmed.ncbi.nlm.nih.gov/8603904/>
- Laforcade AM, Freeman LM, Shaw SP, Brooks MB, Rozanski EA and Rush JE (2003). Hemostatic changes in dogs with naturally occurring sepsis. *Journal of Veterinary Internal Medicine*, 17: 674-679. DOI: <http://dx.10.1111/j.1939-1676.2003.tb02499.x>
- Lamm CG and Rezabek GB (2008). Parvovirus infection in domestic companion animals. *Veterinary Clinics of North America: Small Animal Practice*, 38(4): 837-50. DOI: <http://dx.10.1016/j.cvsm.2008.03.008>
- Kalli I, Leontides LS, Mylonakis ME, Adamama-Moraitou K, Rallis T and Koutinas AF (2010). Factors affecting the occurrence, duration of hospitalization and final outcome in canine parvovirus infection. *Research in Veterinary Science*, 89(2): 174-178. DOI: <http://dx.10.1016/j.rvsc.2010.02.013>
- Kelly WR (1990). *Veterinary Clinical Diagnosis*. 3rd edit. Bailliere Tindall, London. Available at: <https://www.amazon.com/Veterinary-Clinical-Diagnosis-W-R-KELLY/dp/0702009067>
- Khare DS, Gupta DK, Shukla PC, Das G, Tiwari A, Meena NS and Khare R (2019). Prevalence of canine parvovirus infection in dogs in Jabalpur (M.P.). *Journal of Entomology and Zoology Studies*, 7(3): 1495-1498. Available at: <https://www.entomoljournal.com/archives/?year=2019&vol=7&issue=3&ArticleId=5386>
- Marcovich JE, Stucker KM, Carr AH, Harbison CE, Scarlett JM and Parrish CR (2012). Effects of canine parvovirus strain variations on diagnostic test results and clinical management of enteritis in dogs. *Journal of American Veterinary Medical Association*, 241(1): 66-72. DOI: <http://dx.10.2460/javma.241.1.66>
- Martella V, Cavalli A, Decaro N, Elia G, Desario C, Campolo M, Bozzo G, Tarsitano E and Buonavoglia C (2005). Immunogenicity of an intranasally administered modified live canine parvovirus type 2b vaccine in pups with maternally derived antibodies. *Clinical and Diagnostic Laboratory Immunology*, 12(10): 1243-1245. DOI: <http://dx.10.1128/CDLI.12.10.1243-1245.2005>
- McAdaragh JP, Eustis SL, Nelson DT, Stotz I and Kenefick K (1982). Experimental infection of conventional dogs with canine parvovirus. *American Journal of Veterinary Research*, 43: 693-696. Available at: <https://europepmc.org/article/med/7073093>
- McCaw DL and Hoskins JD (2006). Canine viral enteritis. In: Green CE, editor. *Infectious Diseases of the Dog and Cat*. 4th ed. St Louis, MO: Saunders, pp. 63-73. Available at: <https://www.elsevier.com/books/infectious-diseases-of-the-dog-and-cat/sykes/978-1-4160-6130-4>
- Mukhopadhyay HK, Thanislass J, Pillai RM, Antony PX and Rao VN (2012). Detection of canine distemper by nested PCR. *The Indian veterinary journal*, 89(11): 9-11. Available at: <http://www.connectjournals.com/archives.php?bookmark=CJ-000735>
- Mylonakis EM, Kalli I and Rallis ST (2016). Canine parvoviral enteritis: an update on the clinical diagnosis, treatment, and prevention. *Veterinary Medical Research Reports*, 7: 91-100. DOI: <http://dx.10.2147/VMRR.S80971>
- Pollock RVH and Carmichael LE (1988). Canine viral enteritis. In: Barlough JE, editor. *Manual of small animal infectious diseases*. New York: Churchill Livingstone, pp. 101-117. Available at: <https://www.amazon.com/Manual-Small-Animal-Infectious-Diseases/dp/0443085080>

