

**THERAPEUTIC MANAGEMENT OF PARVO VIRUS
INFECTION IN DOGS**

THESIS

Submitted

in partial fulfillment of the requirements for the Degree of

MASTER OF VETERINARY SCIENCE

IN

VETERINARY CLINICAL MEDICINE, ETHICS AND JURISPRUDENCE

BY

**BADWAIK PRENAL YOGESH
Enrolment No: V/14/012**

Nagpur Veterinary College, Nagpur

MAHARASHTRA ANIMAL AND FISHERY SCIENCES

UNIVERSITY, NAGPUR - 440 001

(INDIA)

2022

DECLARATION OF STUDENT

I hereby declare that the experimental research work and interpretation of the thesis entitled “**THERAPEUTIC MANAGEMENT OF PARVO VIRUS INFECTION IN DOGS**” or part thereof has not been submitted for any other degree or diploma of any University, nor the data have been derived from any thesis/publication of any University or scientific organization. The sources of materials used and all assistance received during the course of investigation have been duly acknowledged.

Date:

Signature

(BADWAIK PRENAL YOGESH)

Enrolment No. V/14/012

**Counter signed by
Chairman, Advisory Committee
with date**

DECLARATION OF ADVISORY COMMITTEE

BADWAIK PRENAL YOGESH has satisfactorily prosecuted his course of research for a period of not less than one semester and that the thesis entitled **“THERAPEUTIC MANAGEMENT OF PARVO VIRUS INFECTION IN DOGS”** submitted by him is the result of research work is sufficient to warrant its presentation to the examination in the subject of **Veterinary Clinical Medicine, Ethics and Jurisprudence** for the award of **MASTER OF VETERINARY SCIENCE** degree by the Maharashtra Animal and Fishery Sciences University, Nagpur.

We also certify that the thesis or part thereof has not been previously submitted by him/her for a degree of any other University.

Signature

Place: Nagpur

Date:

(Dr. C. G. Panchbhai)

Chairman/Guide

Assistant Professor

Department of Veterinary Clinical
Medicine, Ethics & Jurisprudence

Advisory Committee

	Name	Designation	Signature
1.	Dr. C. G. Panchbhai (Chairman)	Assistant Professor Dept. of Vet. Clin. Medicine, Ethics & Jurisprudence	_____
2.	Dr. V. M. Dhoot (Member)	Professor & Head Dept. of Vet. Clin. Medicine, Ethics & Jurisprudence	_____
3.	Dr. G. R. Bhojne (Member)	Assistant Professor Dept. of Vet. Clin. Medicine, Ethics & Jurisprudence	_____
4.	Dr. S. V. Upadhye (Member)	Director of Instructions and Dean (Vet), MAFSU, Nagpur	_____
5.	Dr. S. M. Kolangath (Member)	Subject Matter Specialist (Animal Biotechnology) Wildlife Research & Training Centre, Gorewada, Nagpur	_____

CERTIFICATE

This is to certify that the thesis entitled, “**THERAPEUTIC MANAGEMENT OF PARVO VIRUS INFECTION IN DOGS**” submitted by **BADWAIK PRENAL YOGESH** to the Maharashtra Animal and Fishery Sciences University in partial fulfillment of the requirement for the degree of **MASTER OF VETERINARY SCIENCE** has been approved by the Student's Advisory Committee after examination in collaboration with the External Examiner.

Name & signature of
External Examiner

Signature with seal
Head of Department

(Dr. C. G. Panchbhai)
Chairman/Guide
Assistant Professor
Department of Veterinary Clinical
Medicine, Ethics & Jurisprudence.

Advisory Committee

Name	Designation	Signature
1. Dr. C. G. Panchbhai (Chairman)	Assistant Professor Dept. of Vet. Clin. Medicine, Ethics & Jurisprudence	_____
2. Dr. V. M. Dhoot (Member)	Professor & Head Dept. of Vet. Clin. Medicine, Ethics & Jurisprudence	_____
3. Dr. G. R. Bhojne (Member)	Assistant Professor Dept. of Vet. Clin. Medicine, Ethics & Jurisprudence	_____
4. Dr. S. V. Upadhye (Member)	Director of Instructions and Dean (Vet), MAFSU, Nagpur	_____
5. Dr. S. M. Kolangath (Member)	Subject Matter Specialist (Animal Biotechnology) Wildlife Research & Training Centre, Gorewada, Nagpur	_____

Associate Dean
Nagpur Veterinary College,
Nagpur

ACKNOWLEDGEMENT

First of all, I Praise **God**, the Almighty and **my Patients**, for their blessing and providing me the strength to face and conquer the barriers that come on my path.

A formal word of acknowledgment will hardly fulfill the end of justice while expressing my deep sense of gratitude and indebtedness to my guide **Dr. C. G. Panchbhai**, Assistant Professor, Dept. of Veterinary Clinical Medicine Ethics & Jurisprudence, for his noble guidance, keen interest, pragmatic approach, affectionate encouragement, generous help and sustained interest with attention to fine details during my postgraduate studies and research work. And during the most difficult times when writing this thesis, he gave me the moral support and the freedom I needed to move on.

I would like to express my gratitude to **Dr. A. P. Somkuwar**, Associate Dean, Nagpur Veterinary College, for all the support in the successful completion of my research work.

I am very thankful to **Dr. V. M. Dhoot**, Head, Dept. of Veterinary Clinical Medicine, Ethics & Jurisprudence for his benevolent guidance, constructive criticism, liberal attitude, and whole-hearted encouragement. My heartfelt gratitude to **Dr. G. R. Bhojne**, Assistant Professor, Dept. of Veterinary Clinical Medicine, Ethics & Jurisprudence. Sincerely, I deem it a great privilege to work under his guidance and the knowledge he has imparted to me. I thank him for his faith in me. With his sense of caring and humor, he has come to be someone I will always hold in high esteem. I would also like to thank **Dr. Rajendra Ghadge**, Assistant Professor, Department of Veterinary Clinical Medicine, Ethics & Jurisprudence for his support and kind help.

I am very thankful to **Dr. S. V. Upadhye**, Director of Research, TVCC, Nagpur, and members of my advisory committee for his kind co-operation, valuable inputs, timely advice and affection during the entire period of research work and for that I am very grateful.

Dr. Sujit Kolangath, Subject Matter Specialist (Animal Biotechnology), WRTC, Nagpur, has been a never-ending source of support and knowledge sharing. He is the coolest professor I have ever met. His invaluable contribution to my research work and the knowledge he rendered in the limited period will always be remembered. I am grateful for his acquaintance and his support for my PG thesis.

My heartfelt gratitude to all my clinical professors **Dr. S. B. Akhare**, **Dr. Gauri Khante**, **Dr. B. K. Bhadane**, **Dr. Manoj Patil**, **Dr. M. S. Bawaskar** who have been there at TVCC making learning and practicing memorable. I want to express my cordial gratitude to **Dr. Atul Dhok**, Assistant Professor, Department of Animal Nutrition for his kind co-operation, valuable inputs, timely advice, **Shri. D. V. Patil**, Assistant Professor of Statistics and **Shri. S. N. Gawande**, University Librarian, MAFSU, Nagpur, for their unconditional help rendered throughout the course of study.

My sincere thanks to **Dr. Kalyani Thakur**, **Dr. Ushma Patel**, and **Dr. Gurnoor Kaur** for being so supportive, understanding, and sharing their valuable insight throughout the research. I am extremely grateful for my amazing seniors, **Dr. Sameeksha C**, **Dr. Kriti Dohre**, **Dr. Aditya Singh**, **Dr. Sameer Shendre**, **Dr. Ambika Arvind**, **Dr. Tista Joseph**, **Dr. Shital Yesambare**, and **Dr. Sunny Magar** for imparting their knowledge and helping me in the early days of my post-graduation and motivating me to keep learning. My special thanks to my colleagues, who are also my friends **Dr. Ashish**, **Dr. Arvind**, **Dr. Vrushali**, **Dr. Khushboo**, **Dr. Shreelaxmi**, **Dr. Dipali**, **Dr. Kirti**, **Dr. Sujit**, **Dr. Omsatya**, **Dr.**

Maresh, Dr. Ravi and Dr. Prajakta, and Dr. Omkar who were great support and made my life much easier in Nagpur.

*I am fortunate to have **Dr. Shrideeksha** and **Dr. Prashant** as my juniors. My loving thanks to them for being so loving, supportive, entertaining. They have always impressed me with their immense love and dedication toward patients' best care. I will always remember and cherish the moments spent with them. I also thank **Dr. Gunjan, Dr. Ramprasad, Dr. Ketaki, and Dr. Saddam** for being such a helping hand in TVCC and for the fun rapport we share.*

*I am blessed to meet **Dr. Ashish** and **Dr. Arvind**, and immensely grateful for their love, support, and everything. Words cannot express how much you guys mean to me.*

*I am extremely grateful for my dearest friend **Dr. Javed Ashar** who is the one-stop destination for all my problems and has been a great support always. I also thank **Dr. Chetan, Dr. Vivek, Dr. Ujjwal, Dr. Viraj, Dr. Sagar**, Lovely AM Series, wonderful Internship Group members **Dr. Ajay, Dr. Piyush, Dr. Ankush, Dr. Aishwarya, Dr. Kirti**, and my best friend **Dr. Manoj** who were not present physically but were always amazing moral support, also thank to my dear junior **Dr. Divya** and **Vaibhav** for help and joyous company. You all mean a lot to me.*

*Any words would be insufficient to acknowledge **Mrs. Shilpa Deo Mam** for teaching me different laboratory techniques which will help me in improving my patients' health and laboratory work required for my thesis also for unintentional love and support. I am very thankful to **Roshan Dada** and **Tahseen Bhai** for the infinite patience and warmth they always extended towards me. I am grateful to **Shri. Anil Kumre** and all the supporting staff at the TVCC: **Golu Dada, Rupesh Kaka, Shubham Bhau, Nachne Kaka, Chamke Kaka, Sohan Kaka, Meshram Bhau, Lokhande Bhau, Barapatre Kaka, and Jambulkar Kaka** who made each working day smoother and easier.*

*Lastly, I wish to express my eternal indebtedness and heartfelt love to the heroic lady my mother **Sharda Badwaik** and father **Yogesh Badwaik** for their unwavering love, support, encouragement, faith in me, and allowing me to be as ambitious as I wanted. I am heartfelt thanks to my younger brother **Roshan Badwaik** for teaching me stuff related to technologies that helped me a lot, his encouragement, and invaluable moral support also to my dear cousins **Krunal Badwaik, Deveshri Badwaik**, and **All Family members** for always supporting and encouraging me. My wholehearted thank to my four-legged sweetheart **Romi** for teaching me the life lesson to do better for animals' health and society, also the practicality he imparts to my veterinary advice and approach. I am blessed to have these people in my life. It's their whole-hearted love and never-ending moral support which helped in the completion of my research work successfully.*

If I have missed out on mentioning anyone, I thank you for having contributed to this wonderful journey, with great regard. Thank you all!

Date:

Place:

BADWAIK PRENAL YOGESH

TABLE OF CONTENTS

SR NO.	CHAPTERS	PAGE NO.
1.	INTRODUCTION	1-4
2.	REVIEW OF LITERATURE	5-28
3.	MATERIALS AND METHODS	29-39
4.	RESULTS AND DISCUSSION	40-76
5.	SUMMARY AND CONCLUSIONS	77-80
A)	BIBLIOGRAPHY	i-xii
B)	APPENDIX	xiii
C)	VITA	xiv
D)	THESIS ABSTRACT	xv-xvi
E)	प्रबंध सारांश	xvii-xix

LIST OF TABLES

TABLE NO.	TITLE	PAGE NO.
3.1	QIAamp Fast DNA Stool Mini Kit Contents	37
3.2	Composition of the reaction mixture for PCR	38
3.3	Grouping of dogs	39
4.1	Age-wise prevalence of dogs affected with Canine parvovirus infection	42
4.2	Sex-wise prevalence of dogs affected with Canine parvovirus infection	43
4.3	Breed-wise prevalence of dogs affected with canine parvovirus infection	44
4.4	Months-wise prevalence of dogs affected with Canine parvovirus infection	45
4.5	Clinical signs observed in dogs with canine parvovirus infection	49
4.6	Mean Haemoglobin (g/dL) of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	50
4.7	Analysis of variance for Haemoglobin (g/dL) levels in dogs with CPV infection	51
4.8	Mean Total erythrocyte count (million/ μ l) of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	53
4.9	Analysis of variance for Total erythrocyte count (million/ μ l) levels in dogs with CPV infection	53
4.10	Mean Packed cell volume (%) of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	55
4.11	Analysis of variance for Packed cell volume (%) levels in dogs with CPV infection	56
4.12	Mean Total leucocyte count($10^3/\mu$ l) of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	57
4.13	Analysis of variance for Total leucocyte count ($10^3/\mu$ l) levels in dogs with CPV infection	58

4.14	Mean Neutrophils of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	60
4.15	Analysis of variance for Neutrophils levels in dogs with CPV infection	61
4.16	Mean Lymphocytes of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	62
4.17	Analysis of variance for Lymphocytes levels in dogs with CPV infection	62
4.18	Mean Eosinophils of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	64
4.19	Analysis of variance using three-way classification of Eosinophils levels in dogs with CPV infection	65
4.20	Mean monocytes of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	66
4.21	Analysis of variance using three-way classification of monocytes levels in dogs with CPV infection	66
4.22	Mean CK-MB (IU/L) of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	68
4.23	Analysis of variance using three-way classification of CK-MB (IU/L) levels in dogs with CPV infection	69
4.24	Mean Total Protein (g/dl) of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	71
4.25	Analysis of variance for Total Protein (g/dl) levels in dogs with CPV infection	72
4.26	Mean albumin (g/dl) of dogs affected with CPV infection in different treatment groups and age groups at 0 th and 5 th day intervals	73
4.27	Analysis of variance for albumin (g/dl) levels in dogs with CPV infection	73

LIST OF FIGURES

FIGURE NO.	TITLE	PAGE NO.
4.1	Age-wise prevalence of canine parvovirus infection	43
4.2	Sex-wise prevalence of canine parvovirus infection	43
4.3	Breed-wise prevalence of canine parvovirus infection	45
4.4	Months-wise prevalence of canine parvovirus infection	45
4.5	Clinical signs observed in dogs with canine parvovirus infection	49
4.6	Graphical representation of Average Haemoglobin levels of dogs in different treatment groups	51
4.7	Graphical representation of average TEC levels of dogs in different treatment groups	51
4.8	Graphical representation of average PCV levels of dogs in different treatment groups	57
4.9	Graphical representation of Average TLC levels of dogs in different treatment groups	57
4.10	Graphical representation of Average Neutrophils levels of dogs in different treatment groups	63
4.11	Graphical representation of Average Lymphocytes levels of dogs in different treatment groups	63
4.12	Graphical representation of Average Eosinophils levels of dogs in different treatment groups	67
4.13	Graphical representation of Average Monocytes levels of dogs in different treatment groups	67
4.14	Graphical representation of Average CK-MB levels of dogs in different treatment groups	69
4.15	Graphical representation of Average Total Protein levels of dogs in different treatment groups	69
4.16	Graphical representation of Average Albumin levels of dogs in different treatment groups	73
4.17	Graphical representation of Average Recovery time of dogs in different treatment groups	73

LIST OF PLATES

PLATE NO.	TITLE	PAGE NO.
3.1	Rapid Antigen Test Kit	30
3.2	Collection of faecal sample by using sterile swab	30
3.3	Mixing of swab in buffer	30
3.4	Adding of mixed sample into sample hole	30
3.5	Cardiac Troponin I Rapid Test Kit	30
4.1	Rapid Antigen Test Kit showing positive result	41
4.2	Rapid Antigen Test Kit showing negative result	41
4.3	Agarose Gel Electrophoresis: 1% agarose gel electrophoresis with ethidium bromide revealed amplification of 681 bp as reported by Sheikh et al.,2017	41
4.4	Pale mucous membrane	47
4.5	Congested mucous membrane	47
4.6	Haemorrhagic diarrhoea of watery consistency	47
4.7	Whitish and frothy vomitus of CPV infected dog	47
4.8	Depressed and lethargic dog	49
4.9	Dog showing severe dehydration	49
4.10	Cardiac Troponin I Rapid Test showing negative results	75
4.11	Infected dogs before treatment	76
4.12	Recovered dogs after treatment	76

ABBREVIATIONS

%	:	Percentage
α	:	Alpha
@	:	At the rate of
\leq	:	Less than or equal to
\geq	:	More than or equal to
\pm	:	Plus-Minus
<	:	Less than
>	:	More than
®	:	Registered trademark
°C	:	Degree Celsius
°F	:	Degree Fahrenheit
i.e.	:	That is
ALT	:	Alanine transaminase
bp	:	Base pairs
BUN	:	Blood urea nitrogen
B.W.	:	Body Weight
b.i.d	:	<i>bis in die</i>
CAV	:	<i>Canine adeno virus</i>
CBC	:	Complete blood count
CCoV	:	Canine corona virus
C.D.	:	Critical Difference
CK-MB	:	creatinine kinase myocardial isoenzyme
Cl ⁻	:	Chloride
CPV	:	Canine parvovirus
cumm	:	Cubic millimetre
DLC	:	Differential leucocyte count
DNA	:	Deoxyribonucleic acid
DNS	:	Dextrose normal saline
EDTA	:	Ethylene diamine tetra-acetate
EIA	:	Enzyme immunoassay
ELISA	:	Enzyme linked immunosorbent assay
EM	:	electron microscopy
<i>et al.</i>	:	Et alia (and others)

e.g.	:	Example
FAT	:	Fluorescent antibody test
gm	:	Grams
g/dL	:	Gram per decilitre
HA	:	Haemagglutination
Hb	:	Haemoglobin
HI	:	Haemagglutination inhibition
hr	:	Hours
IC	:	Immunochromatography
IU/kg	:	International Unit per Kilograms
IM	:	Intramuscular
IV	:	Intravenous
K ⁺	:	Potassium
kg	:	Kilograms
mcg	:	Microgram
MCHC	:	Mean corpuscular haemoglobin concentration
MCV	:	Mean corpuscular volume
mins	:	Minutes
ml	:	Millilitre
ml/kg/d	:	Millilitre per kilogram per day
mmol/l	:	Millimole per litre
Na ⁺	:	Sodium
NS	:	Non-Significant
No.	:	Numbers
n-PCR	:	Nested polymerase chain reaction
PCR	:	Polymerase chain reaction
PCV	:	Packed Cell Volume
Pvt. Ltd.	:	Private Limited
pH	:	potential of Hydrogen
P.O.	:	Per Os
RBC	:	Red Blood Cells
RL	:	Ringer's Lactate
S/C	:	Subcutaneous
sec	:	Second
SGOT	:	Serum glutamic oxaloacetic transaminase
SGPT	:	Serum glutamic pyruvic transaminase

S.E	:	Standard error
S.C.	:	Subcutaneous
S.D.	:	Standard Deviation
TEC	:	Total Erythrocyte Count
TID	:	Thrice a day
TLC	:	Total Leucocyte Count
VCC	:	Veterinary Clinical Complex
viz.	:	Namely
WBC	:	White Blood Cell
WASP	:	Web Agri Stat Package
VP2	:	Viral Protein 2

INTRODUCTION

Having a pet is always a joy as the pets are one of the most faithful family members and loyal companions. Dogs, like humans, are highly sociable animals, and this behavioral resemblance explains their trainability, playfulness and ability to adapt to human families and social circumstances. In many countries, the most common and perhaps the most significant role of dogs is as companions. Dogs play a variety of roles in human society. The list is not short; however, some essential roles include service and assistance work, therapy, search and rescue, herding, guarding, police work and alerting owners regarding medical problems. The recent coronavirus outbreak has harmed our daily routine, as well as our physical and emotional health. In this challenging situation, to ease loneliness and boredom, our furry friend has emerged as the ray of hope in our lives in the last two years. Dogs have served humans in so many ways that they have earned the title of "man's best friend."

Considering the significance of dogs in our life, it is our responsibility to take care of their health. Dogs, in general, suffer from numerous health issues, which include diseases caused by a virus, bacteria, fungi, protozoa, etc. Among these, viral gastroenteritis has become a paramount cause of morbidity and mortality in dogs, with Canine Parvovirus (CPV) being the lethal virus that causes gastroenteritis in dogs.

In 1978, the canine parvovirus (CPV) was initially identified as a clinical entity that causes enteritis in dogs (Esfandiari and Klingeborne, 2000). Canine parvovirus enteritis is one of the highly contagious and fatal diseases in dogs, affecting primarily young and unvaccinated dogs, exhibiting acute infectious gastrointestinal illness with high morbidity (100%) and mortality rate as high as 91% in the absence of treatment (Prittie 2004; Merck 2010 and Aiello *et al.*, 2016). Canine parvovirus enteritis is caused by canine parvovirus type-2 (CPV-2). Canine parvovirus is a member of the genus Parvovirus and the family *Parvoviridae*. It has a small, non-enveloped, single-stranded negative-sense DNA genome with a size of 5.2 kilobytes. Parvovirus was first recognized as a cause of

gastrointestinal and respiratory ailment in dogs in 1967 and was designated CPV-1, or canine minute virus. Later, in 1978, in the United States, outbreaks of an unknown infectious gastrointestinal disease were documented, which was caused by new species of the genus Parvoviridae; the virus was named CPV-2 to differentiate it from CPV-1 (Nandi and Goddard, 2010). The CPV-2 infections have emerged as a problem in dogs in recent times around the world and are responsible for classic parvoviral enteritis; and has three strains named CPV-2 a, b, and c detectable only by monoclonal antibodies and genetic analysis (Nelson & Couto, 2013 and Odueko, 2020). The virus is resistant to several common detergents, disinfectants, as well as temperature, pH fluctuations and persist in soil or on fomite for months to years (Aiello *et al.*, 2016).

Canine parvovirus infection spreads very quickly to susceptible puppies. CPV2 is easily spread by contaminated things such as utensils, shoes and fomites. Feces contaminated places like veterinary hospitals, pet shops and kennels can proficiently spread the virus to susceptible dogs (Khatri, 2017). The disease has a quick clinical course, with death may occurring within 2–3 days of the onset of symptoms (Vaishali & Jain, 2020). CPV-2 causes symptoms 5 to 12 days after infection via the fecal-oral route and preferentially invades and kills quickly proliferating cells (i.e., bone marrow progenitors, intestinal crypt epithelium), (Nelson & Couto, 2013).

The infection has two clinical forms as acute hemorrhagic enteritis and myocarditis. Acute enteritis form is seen chiefly in puppies up to 12 months of age. Anorexia, fever, depression and lethargy are some of the non-specific initial clinical signs whereas typical symptoms include depression, vomiting and small bowel hemorrhagic diarrhea. Myocarditis form can develop from infection in utero or in puppies less than eight weeks old. In this form, the infected puppies could die within 24 hours without showing any signs (Cenk ER & Ok, 2015). Intestinal tract damage caused by viral infection raises the risk of bacterial translocation and coliform septicemia, leading to a systemic inflammatory response which ultimately led to septic shock and death. (Goddard & Leisewitz, 2010). Dehydration and hypovolemic shock often develop quickly due to a large

amount of protein and fluid loss from the gastrointestinal tract (Ahmad & Waheed, 2020).

The incidence of canine parvovirus infection was reported from various continents, viz., Asia, Africa, Australia, Europe and America (Nandi & Kumar, 2010). Animal shelters, pet stores, and breeding kennels have a higher incidence. Canines of any age can be infected with CPV but puppies from the age group of six weeks to four months are the most vulnerable to infection. Breeds like Doberman Pinschers, Rottweilers, German Shepherds, American Pit Bull Terriers, English Springer Spaniels, etc. are at higher risk infection (Nandi & Kumar, 2010). Factors that predispose parvovirus infection in puppies are lack of protective immunity, overcrowding of animals in a small space, parasitism, weaning, stressful and unhygienic environmental conditions (Dash *et al.*, 2019), which makes the diagnosis and management of this disease so critical.

The diagnosis of CPV-2 is based on signalment, history, clinical signs and confirmation by different methods used for detection of this virus, which includes electron microscopy (EM), virus isolation (VI), hemagglutination (HA), ELISA, latex agglutination (LA), in situ hybridization (ISH) and polymerase chain reaction (PCR) analysis. Nowadays, the Rapid antigen test kit has shown to be very useful diagnostic tool in screening dogs for CPV infection and is a reliable alternative for diagnosing CPV both in conventional laboratories and remote areas without laboratories (Shima *et al.*, 2021). Any young puppies aged between six weeks to six months with or without an appropriate immunization history that show indicators of enteritis disease should be suspected of CPE and should be treated as soon as possible.

In canine parvovirus infection, if no treatment is given, the survival rate can be as low as 9%, but in tertiary care facilities, it can be as high as 80%. Therefore, therapeutic management of canine parvovirus infection is very crucial, which mainly include fluid and electrolyte therapy along with antibiotics, antiemetics, antacid, astringent and in cases with severe hypoproteinemia, colloids (plasma or synthetic colloids), etc. may be given. In last few years researchers

also studied use of Recombinant feline interferon- ω (rFeIFN- ω), recombinant human granulocyte colony- stimulating factor (G-CSF), recombinant canine G-CSF (rcG-CSF), Equine endotoxin antiserum, immunomodulators, etc for treatment of CPV, however, evidence-based justification for their use is currently lacking. As there is no specific treatment for CPV-2, the best mean of controlling the infection is proper vaccination (Gamage *et al.*, 2020). As the disease is of viral origin, antibiotics will not prevent the virus from multiplying and affecting the host tissues. However, antibiotics are important in the prevention of secondary bacterial infections. Nevertheless, intermittent attempts have been made by researchers and practicing veterinarians to introduce antiviral agents in the therapy. "Oseltamivir" the antiviral drug which was first designed to treat influenza in humans, is now being tried in dogs to treat parvoviral infection. Although several anecdotal reports have come out from practicing veterinarians, especially from North America, indicating its beneficial effects; yet very few studies have been conducted. Hence, more systematic and scientific studies are required to be undertaken to come out with a definite conclusion. For that reason, the present study was designed with the following objectives.

11. OBJECTIVES

1. To record the prevalence of canine parvovirus infection in dogs.
2. To study the diagnosis and comparative therapeutic efficacy of different drugs in canine parvovirus infection in dogs.

REVIEW OF LITERATURE

Some of the representative studies with their significance in the present study entitled “**Therapeutic Management of Parvo Virus Infection in Dogs**” have been reviewed and presented under different sub headings as follow.

2.1 Etiology

Appel *et al.* (1979) reported canine parvovirus in faecal samples of dog population suffering from haemorrhagic enteritis by staining with FAT reagent or haemagglutination test using pig or rhesus monkey red blood cells.

Pereira *et al.* (2000) performed molecular typing on 38 CPV isolates which were collected between 1982 and 1995, and reported that the relative incidence of CPV antigenic types altered over time. CPV-2a was the most common strain identified in 1980, but from 1990 to 1995, it was largely supplanted by CPV-2b.

Cavalli *et al.* (2001) applied a hemagglutination assay to discover a CPV-2b strain in the faeces of an adult vaccinated dog with acute gastroenteritis and found that a CPV-2b-like pattern.

Decaro *et al.* (2007) investigated the tissue distribution of canine parvovirus type 2 antigenic variations in dogs in Italy. In their investigation, they included 12 dogs who died as a result of canine parvovirus infection. CPV was found in all tissues tested using a real-time PCR assay, with the highest levels in lymphoid tissue and the lowest levels in the urinary tract. Surprisingly, a significant amount of CPV nucleic acid was discovered in nerve tissue.

Parthiban *et al.* (2010) examined 128 faecal samples collected from diarrheic dogs during March 2007 and February 2008 by PCR assay and they revealed 53.1% of the samples positive for CPV.

Bhat (2012) reported occurrences of gastroenteritis in dogs in the Himachal Pradesh region of Palampur. PCR was used to test 45 faecal samples for CPV infection, out of which 18 samples were found positive for CPV-2 DNA.

Zhao *et al.* (2013) analysed a circulating canine parvovirus's genotypic and pathobiological characteristics in China. They examined 31 samples and found 29 positive for CPV-2a and two positive for CPV-2b.

Ogbu *et al.* (2017) reported that canine parvovirus (CPV) is a single-stranded non-enveloped DNA virus belonging to the *parvoviridae* family that replicates in quickly proliferating cells. The virus is exceedingly hardy, surviving exposure to many common disinfectants and persisting in soil or on fomites for months to years. They stated that Canine parvovirus is currently divided into three strains: CPV-2a, CPV-2b, and CPV-2c.

Sheikh *et al.* (2017) observed that CPV-2b is currently the only virus circulating in Sulaimani/Iraq and the VP2 encodes a viral capsid protein that is the major structural protein of CPV-2 which is involved in the host immune response. They concluded, a small number of mutations may result in increased pathogenicity.

Chethan *et al.* (2021) screened a total of 216 faecal samples during the study period, out of which 90 (41.67%), 12 (5.56%), and 26 (12.04%) samples were found to be positive for CPV-2, CAV-1, and co-infection, respectively. The analysis of positive samples revealed that the occurrence of CPV-2 and CAV-1 infection was highest in young dogs of age group 0-3 months (CPV-2=44.44% and CAV-1=33.33%)

2.2 Prevalence

Archana *et al.* (2009) reported that the prevalence of canine parvovirus infection was 45.30% (58/128). The age-wise maximum prevalence (70.20%) was found in dogs up to 6 months, the breed-wise maximum prevalence (56.90%) was in non-descript dogs, whereas sex-wise prevalence revealed no significant difference in males (45.9%) and females (43.3%).

Kumar *et al.* (2011) observed that among the different breeds of dogs, German shepherds were found to be highly susceptible (69.2%), followed by non-descript breeds (63.1%), with overall positivity was 59.5%.

Parthiban *et al.* (2010) examined 128 faecal samples of diarrheic dogs, and reported that incidence was very high in young puppies of 0-6 months of age (83.8%), in the summer season (60.3%), in males (67.6%), and in non-vaccinated dogs (79.4%). The incidence of CPV infection was 20.6% in vaccinated dogs.

Singh *et al.* (2013) found that dogs of the age group <6 of months were more susceptible in comparison to of >6 months of age, and the highest occurrence was noted in unvaccinated dogs and dogs in cohabitation with other dogs. Breed wise distribution of CPV in dogs revealed highest prevalence in Doberman (77.78%), followed by Spitz (78.57%), German shepherd (70.00%), Labrador (58.79%) and Pomeranian (45.4%). They reported that CPV was prevalent in the Mathura and nearby area and it was more common in pups of age less than 6 months old and more prevalent in German shepherd, Labrador and Pomeranian breeds of dog.

Nahat *et al.* (2015) observed that prevalence of parvovirus infection was found to be significantly higher in 6 months of age (58.3%) than those >24 months of age. The prevalence of canine parvovirus infection was non-significantly higher in males (34.4%) than in females (30.2%).

Reddy *et al.* (2015) screened 217 faecal samples of dog, out of that 72 samples were positive for CPV infection with an overall prevalence of 33.17 percent. Breed-wise analysis indicated the highest prevalence (43.75%) in Spitz and the least prevalence (28.71%) in Mongrel, sex-wise data analysis revealed a higher prevalence (37.30%) in male dogs than female (27.47%) dogs, month-wise prevalence of CPV infection revealed the highest prevalence (48.97%) in July and the lowest (20%) in March. Further they stated that the most concerning age is 0-6 months and least is above 1year old dogs. CPV infection prevalence was higher (35.41%) in un-vaccinated dogs than the vaccinated dogs (16.00%).

Hasan et al. (2017) reported 57.4% infection in 1-3 months of age and 28.9% in 4-6 months of age and 13.2% among vaccinated and 64.4% in non-vaccinated dogs. Significant ($P < 0.05$) variation in prevalence of CPV also observed in different breeds viz. indigenous (50.0%), Spitz (28.2%), Lhasa (18.1%), Doberman (40.0%) and German Shepherd (46.6%).

Mehta et al. (2017) screened faecal samples of 145 dogs, out of which 63 samples were positive with an overall prevalence of 43.44%. Breed wise analysis of data indicated higher prevalence in indigenous breeds (46.67%) than the exotic breeds (42.00%). CPV infection in relation to age was highest (54.05%) in 0-2 months and least (30.43%) in 4-6 months old dogs. Sex wise analysis revealed the higher prevalence in female dogs (48.15%) than male (40.66%). Month wise prevalence of CPV infection was found highest in February (65.00%) and lowest in June (12.50%). The prevalence of CPV infection was high (44.03%) in unvaccinated dogs than the vaccinated dogs (36.36%).

Khare et al. (2019) reported that dogs between 0-3 months of age showed the highest prevalence 11.9%, followed by 3-6 months of age 7.09%, and 6-12 months of age 5.31%, respectively. In contrast, the lowest prevalence of canine parvovirus was reported in dogs above 12 months of age. The sex-wise prevalence revealed a higher prevalence in males (7.91%) than females (6.36%). The maximum prevalence was noticed in non-descript dogs, i.e., 12.57%, followed by Great Dane, German shepherd, Spitz, Dalmatian, Labrador, Doberman, and Lhasa apso in which prevalence was found as 10%, 6.12%, 5.26, 3.82%, 3.61%, 3.57%, and 2.77% respectively. During that study, the dogs reared on a vegetarian diet showed significantly higher prevalence, 8.90%, than dogs reared on a non-vegetarian diet, 3.38%.

Sharma et al. (2019) studied 102 faecal samples of dogs by haemagglutination assay, from which they found 40.19% positive. Age wise prevalence was (69.23%) in (0-6 month) age group which is more than (7-12 month) (13.33%) and more than a year group (5%). Sex wise prevalence was more in males (47.94%) than in females (20.78%). Breed wise prevalence was

more in Labrador (78.57%) followed by Doberman Pinscher (62.50%) lower prevalence was reported in Pomeranian and German shepherd breeds. Non vaccinated canines showed a high prevalence of (42.70%) in contrast to vaccinated canines where no disease prevalence was detected. In season wise prevalence, spring season showed more prevalence (54.76%) followed by summer season (37.5%) and the least prevalence of (11.11%) was recorded in the winter season.

Alves (2020) carried out retrospective study of 103 dogs infected naturally with parvovirus CPV-2 and noticed that the mean age of animals diagnosed with canine parvovirus was 5.7 ± 0.7 months, with 2/3 of cases (66.7%) concentrating within three to six months of age also this age group accounted for 42.8% (12/28) of deaths in his study. Out of 103 animals affected by canine parvoviruses, unvaccinated (46.6%, n=48) or had an incomplete vaccination schedule (30.0%, n=31). Among the other dogs, 4.9% (n=5) had complete vaccination, and in 18.4% (n=19), there was no information about vaccination status.

Horecka et al. (2020) observed a distinct parvovirus season peaking in May and June and troughing in August, September, December, and January, which contributed as much as 41 animals peak-to-trough in the monthly population (with a potential, smaller season occurring in October), also low-weight and male animals were at higher risk for death, whereas age was not a significant contributing factor.

Suvethika and Kumar (2021) stated that CPV is a highly contagious disease that primarily affects puppies aged 1-6 months.

Tanwar et al. (2020) evaluated breed wise prevalence, and found that CPV infection was more prevalent in Labrador 41.66% (10 out of 24), followed by Pitbull 40% (8 out of 20), Rottweiler 33.33% (2 out of 6), German Shepherd 30% (6 out of 20), non-descriptive 16.66% (2 out of 12), Dalmatian 12.50% (1 out of 8) and Spitz 10% (1 out of 10) breed of dogs. Breed-wise comparison indicated that Labrador and Pitbull breeds of dogs were mainly affected with CPV.

Venn *et al.* (2020) recorded breed wise prevalence in CPV infection, which included Chihuahua (9/40, 22.5%), unknown mixed breed (8/40, 20%), Pit Bull (5/40, 12.5%), Miniature Poodle (3/40, 7.5%), Pug (2/40, 5%), Maltese (2/40, 5%), Labrador Retriever (2/40, 5%), and one each of Border Collie, Australian Heeler, Boxer, Miniature Pinscher, Great Dane, Standard Poodle, and Siberian Husky (1/40, 2.5%).

Chethan *et al.* (2021) discovered that the prevalence of CPV-2 infection was higher in female dogs (51.11%) than male dogs (48.88 %); there was no similar difference in the prevalence of CAV-1 infection. The occurrence of CPV-2 infection was found highest in the Labrador Retriever breed (35.56%), and none of the faecal samples of Cocker Spaniel breed were found positive. The occurrence of co-infection with CPV-2 and CAV-1 was found to be highest in the non-descript breed (30.77%). The occurrence of CPV-2, CAV-1, and co-infection with both the viruses was highest in unvaccinated dogs and was least in the dogs that received three doses of vaccine.

Mahaprabhu *et al.* (2021) investigated that the prevalence of canine parvoviral enteritis was higher in vaccinated exotic breed puppies (90%) than vaccinated native breeds (40%). The age-wise prevalence of canine parvoviral enteritis showed no difference in both age groups below two months and above two months, whereas no significant difference was noticed in both sexes in vaccinated and unvaccinated puppies. The vaccinated puppies showed a higher incidence as well as recovery rate than unvaccinated puppies.

2.3 Diagnosis

Goddard *et al.* (2008) confirmed the diagnosis of CPV in 62 puppies between the ages of 6 and 24 weeks by faecal transmission electron microscopy (EM) within 24 hours.

Sagar *et al.* (2008) studied canine parvoviral infection by screening 27 dogs based on clinical signs like vomition, blood, and fetid diarrhoea and out of which diagnosis of 18 dogs was confirmed by polymerase chain reaction (PCR).

Archana *et al.* (2009) collected a total of 128 faecal samples of dogs suspected of canine parvovirus infection and were examined by Haemagglutination (HA) and Haemagglutination inhibition (HI) tests, out of which 58 dogs were found positive.

Kumar *et al.* (2010) implemented nested polymerase chain reaction (n-PCR), developed using published pCPV-2ab as external primer set along with self-designed and custom synthesized pCPV-IN as internal primer set for the detection of canine parvovirus in faecal samples of dogs. Out of 52 faecal samples, 27 and 31 were tested positive by one-step PCR and n-PCR, respectively.

Pillai and Deepa (2010) detected CPV by using the canine parvovirus antigen rapid test kit and Haemagglutination test. The confirmatory diagnosis of CPV was done on basis of titer value 80 by haemagglutination test.

Castro *et al.* (2013) screened 50 samples of dogs less than one year of age for CPV by using a commercial Enzyme immunoassay (EIA) based Canine Parvovirus Antigen Test (SNAP). Viral DNA was extracted from 10% faecal suspensions in Tris-Ca²⁺ (0.01 M, pH 7.2) using phenol/chloroform/isoamyl alcohol (Invitrogen) and silica/ guanidine thiocyanate. Polymerase chain reaction with the 555For/555Rev primers (4003–4585) was performed to amplify a 583 bp fragment of the VP2 gene. After an initial incubation at 94°C for 10 min, 40 cycles of amplification were carried out at 94°C for 30 s; 50°C for 1 min, and 72°C for 1 min, followed by a final extension at 72°C for 10 min. Sequencing of the amplicon was performed using BigDye terminator v.1.1 cycle sequencing kit, and the analysis of the residues 426 (nt 4062–64), 555 (nt 4449–51), and 570 (nt 4494) confirmed the infection by wild type CPV strain in puppies that received ML CPV vaccine. Out of 50 dogs, 13 dogs were confirmed as positive for CPV by PCR.

Singh *et al.* (2013) studied canine faecal samples from 100 dogs which were showing the clinical signs of gastroenteritis in and around Mathura, Uttar Pradesh, India. DNA was extracted by the phenol-chloroform method. CPV

vaccine strain was used as a positive control. Polymerase Chain Reaction (PCR) was carried out to amplify VII/VPS gene using a set of 20-mer primers (pCPV-RT (Forward): 5-CAT TOG GOT TAC CAC CAT TT-3: (Reverse) 5-C0A ACC TCA OCT GOT CTC AT3] from position 3136-3156 to 3276-3295 of VP1/P2n A PCR product of approximately 160 bp was generated with positive faecal samples and CPV vaccine strain.

Salem (2014) used immunochromatographic assay for CPV antigen detection using commercial test kits (Canine Parvo Virus Ag Rapid Test Kit by Immunochromatographic Assay) and found 24 positive dogs out of 35. Further the causative organism was confirmed by RT-PCR.

Sulthana (2015) observed 17 dogs, exhibiting signs of vomiting and foul-smelling bloody diarrhoea, which were screened for parvovirus infection by a polymerase chain reaction from faecal samples. PCR was carried out using primer pair 555 forward- 5'-CA GG AA GA TATCC AG AAGGA-3' and 555 reverse- 5'- G G T G C T A G T T G A T A T G T A A T A A A C A - 3' in which a band of 583 bp was obtained in 12 samples.

Tinky *et al.* (2015) detected the presence of canine parvovirus (CPV) in faecal samples of diarrhoeic dogs by conventional polymerase chain reaction (PCR). The sensitivity of Rapid CPV Ag test kit was compared with PCR, and they found that out of 22 samples, 6 samples were detected negative with Rapid CPV Ag test kit which were found positive by PCR.

Parthiban *et al.* (2016) confirmed twenty CPV cases of non-descript pups in the age group of 6-8 weeks by PCR (using primer pair H_{for}/H_{rev}).

Ford *et al.* (2017) performed a retrospective study in which DNA was extracted from formalin-fixed, paraffin-embedded tissues from 40 cases and 41 control dogs under 2 years of age from 2007 to 2015. Nucleic acid was extracted from two 10-mm-thick FFPE tissue sections using the QIAamp DNA FFPE Tissue kit (QIAGEN, Valencia, CA). Conventional PCR was performed using house-keeping gene RPS19 primers and parvoviral VP1 to VP2 primers.

Conventional PCR using VP1/VP2 PV primers resulted in amplification of the expected 186-bp product in 12 of the 40 cases and 2 of the 41 controls. CPV-2 VP2 messenger RNA (mRNA) was amplified by RT-qPCR in 12 of the 12 cases and 2 controls having amplicon sequences matching banked CPV-2 with a reaction amplification efficiency of 97.9%. Based on their finding they conclude that, despite of vaccination, CPV-2 remains an important cause of myocardial damage in dogs.

Sheikh *et al.* (2017) analysed eight faecal samples of unvaccinated dogs with signs of vomiting and bloody diarrhoea during the period from December 2013 to May 2014 in different locations in Sulaimani Kurdistan, Iraq. After PCR detection, they found that all viral sequences were CPV-2b variants, which differed genetically by 0.8% to 3.6% from five commercially available vaccines and alignment between eight nucleotides of field virus sequences showed 95% to 99.5% similarity.

Khare *et al.* (2020) reported canine parvovirus infection in thirty-two cases of dogs suffering from vomition, diarrhoea and depression. The confirmatory diagnosis was carried out by Scanvet Parvo test kit, which is a immunochromatographic assay based kit.

Chethan *et al.* (2021) performed the screening of CPV-2 and CAV-1 in faecal samples by direct detection of viral genome by polymerase chain reaction (PCR) based on VP2 gene and E3 gene of CPV-2 and CAV-1, respectively. The polymerase chain reaction (PCR) for the identification of CPV-2 was carried out by using primers (FP- TGATTGTAAACCATGTAGACTA and RP-TAAGTCAGTATC AAATTCTTTATC) for targeting the VP2 gene and the identification of CAV-1 was made by using primers (FP-CGCGCTGAACATTACTACCTTGTC and RP-CCTAGAGCACTTCGTGTCCGCTT) specific to E3 gene as evident by the presence of expected 631 bp and 508 bp product in an agarose gel under UV transillumination, respectively.

Munoz *et al.* (2021) studied eighteen puppies with CPE from five veterinary hospitals in the metropolitan area of the Valley of Mexico between March and September 2019. To extract the genetic material, 500 µL DNAzol (Invitrogen®, Massachusetts, USA) and 8 µL Proteinase K (Bioline, London, UK) were added at a concentration of 60 µg/mL to the stored sample (500 µL) at – 75 °C. The mixture was incubated at 56 °C for 30 min, and DNA was extracted according to the DNA zol reagent protocol, using 100 and 75% molecular biology grade ethanol (Sigma Aldrich, Darmstadt, Germany). The precipitate was hydrated with 30 µL DNase-free water, and DNA was quantified by spectrophotometry in a Nano Drop spectrophotometer. The end-point PCR was carried out following the protocol of Master Mix Platinum II PCR MM (Invitrogen®, Massachusetts, USA) in a MultiGene® Optimax thermal cycler with an initial denaturation temperature of 95 °C for 10 min and subsequently, 30 cycles of denaturation at 95 °C for 30 s, annealing at 55 °C for 30 s, and extension at 72 °C for 30 s; the total time of the PCR was 2 h and 30 min. The primers used forward 5'-GAC CAG CTG AGG TTG GTT ATA G- 3' and reversed 5'- GGT GCA TTT ACA TGA AGT CTT GG 3' were directed to the gene coding for the capsid protein (VP2) of CPV-2 and created a 466-bp amplicon.

2.4 Clinical signs

Banja *et al.* (2002) reported that haemorrhagic diarrhoea and vomition were the most common clinical symptoms, however inappetence, haematemesis, fever, and dehydration were found in both single (CPV) and combined infections (CPV and CCV).

Hoskins (2006) observed that vomition is common in CPV-2-infected dogs, followed by diarrhoea, anorexia, dehydration, and an elevated rectal temperature (104-105.8°F). He further stated that the faeces were yellow-grey in colour and were stained or darkened by blood.

Goddard *et al.* (2008) observed lethargy, anorexia, vomiting, hemorrhagic diarrhoea, dehydration, collapse as typical signs in CPV infected puppies between the ages of 6 and 24 weeks, of any breed and either sex.

Sagar *et al.* (2008) observed that high body temperature was the most common symptom in dogs with parvovirus infection, followed by vomiting and finally severe fetid diarrhoea with frank bleeding and dehydration.

Singh *et al.* (2011) noticed clinical signs of CPV in 50 dogs which include increased body temperature (104-106⁰F), increased respiration rate, tachycardia, paleness of mucous membrane of eye, anorexia, diarrhoea, vomition along with marked dehydration. The diarrheic faeces were generally mucoid, yellowish-grey, or blood mixed. The vomitus was greyish watery in nature.

Decaro and Buonavoglia (2012) reported that clinical indications of CPV enteritis appeared after 3–7 days of incubation period and which includes depression, anorexia, vomiting, mucoid or bloody diarrhoea, recurrent dehydration, and fever.

Bastan *et al.* (2013) evaluated 38 puppies with CPV infection and found that puppies with CPV had cardinal clinical signs like anorexia, lethargy, bloody diarrhoea, vomiting, moderate to severe dehydration, tachycardia, and hypothermia.

Castro *et al.* (2013) observed clinical signs such as vomiting, anorexia, lethargy, and haemorrhagic fluid diarrhoea in both CPV and CCoV infected as well as in non-infected puppies and the association of these signs with CPV infection was statistically significant ($P < 0.01$).

Salem (2014) observed the most consistent clinical signs of CPV-infected dogs to have fever, bloody diarrhoea, vomiting and dehydration while the most consistent clinical signs observed in CDV-infected dogs were fever, diarrhoea, respiratory signs, vomiting and dehydration.

ER and OK (2015) reported anorexia (27 case), lethargy (27 case), depression (27 case), fever (10 case $>39.5^{\circ}\text{C}$), vomiting (23 case) and haemorrhagic diarrhoea (21 case) in twenty-seven dogs with parvoviral enteritis.

Kumar and Kumar (2017) found that dogs infected with CPV infection were having prominent clinical signs of anorexia, dullness and vomiting, which was either haemorrhagic (44.55%) or non-haemorrhagic (55.45%). Increased body temperature (104-106°F) was noted in 24.54%, while 75.45% of dogs had a subnormal temperature (98-100°F). The increased body temperature may suggest viremia in the early stage of the disease, while the late reported case had subnormal body temperature due to severe fluid and electrolyte losses.

Ogbu *et al.* (2017) reported that this disease is characterized by lethargy, dehydration, anorexia, fever, vomiting, and diarrhoea that may contain mucus or blood and has a strong unpleasant odour.

Alves (2020) observed that diseased animals had significantly higher median values for heart rates and respiratory rates, however, significantly lower median values for systolic blood pressure compared to those who survived. There was no significant difference in rectal temperature between the survivals and non-survivals. Tachycardia and hypotension may be attributed to decreased tissue perfusion generated by dehydration, secondary to gastrointestinal loss of fluid and proteins.

Gulersoy *et al.* (2020) noticed clinical signs in 30 dogs with parvoviral enteritis, which includes anorexia, stagnation, depression, fever, tachypnoea, tachycardia, lethargy, vomiting, dehydration, elongation at capillary filling time along with tachycardia, haemorrhagic diarrhoea, and shock etc.

Horecka *et al.* (2020) observed inappetence, vomiting, ill formed stool, diarrhoea or bloody diarrhoea, pale gum colour and lethargy or coma. Out of 574 animals that were symptomatic of CPV was estimated to be 230/574 (40%). Out of the 230 dogs with severe symptoms, 70 (30.4%) did not survived.

Kumar *et al.* (2020) reported that clinical examination of CPV-affected dogs showed vomiting, anorexia, haemorrhagic diarrhoea, paleness of mucous membrane, dullness, depression, and restlessness. Also, heart rate and respiration rate were found significantly higher than that of the healthy control group.

Suvethika and Kumar (2021) observed the clinical signs of infection, which include loss of appetite, vomiting, dehydration, foul-smelling bloody diarrhoea, and pale mucous membranes in infected dogs.

Rabbani *et al.* (2021) noted that in the early phases of the disease, dogs experience inappetence, depression, pyrexia, vomiting, and diarrhoea. As the animal continues to lose fluids, body temperature gradually depreciates to subnormal levels. Whereas, after progression of disease following third day of clinical elucidation are highly variable due to differences in viral exposure. Animals exhibiting cardiac signs do not have diarrhoea, as the virus proliferates in muscles of the heart as opposed to intestinal villi. Canine parvovirus can also manifest itself clinically as cardiac myopathy and myocarditis in puppies younger than three months.

2.5 Haematology

Robinson *et al.* (1980) investigated that, in CPV-infected dogs virus causes suppression of the bone marrow, as well as changes in erythroid, myeloid, and megakaryocyte, can result in a drop in RBCs, HB, and PCV levels.

Nappert *et al.* (2002) studied 25 puppies naturally infected with canine parvovirus infection and recorded increase in mean packed cell volume of $41.27 \pm 10\%$.

Goddard *et al.* (2008) noticed severe leukopenia, lymphopenia, monocytopenia, and eosinopenia, as well as the absence of a left shift, which can be indicative of poor prognosis.

Sagar *et al.* (2008) observed that blood picture of CPV affected dogs showed neutrophilia and lymphopenia, however, there were no significant differences in eosinophil, monocyte and basophil percentage. The affected dogs showed lower mean values of haemoglobin, packed cell volume, total erythrocyte count and total leucocyte count.

Roy et al. (2010) reported reduction in haemoglobin, PCV, TEC values along with neutrophilia, lymphopenia, whereas no changes were observed in eosinophils and monocytes in CPV affected groups on day of presentation.

Decaro and Buonavoglia (2012) found that leukopenia (2000-3000 cells/microlitre of blood) as a common finding in CPV enteritis, and normal WBC ranges were occasionally observed, which could be due to concomitant virus-induced lymphopenia and neutrophilia caused by opportunistic bacterial infections.

Bastan et al. (2013) reported that non-survived puppies had lower mean leucocyte, lymphocyte, monocyte, and granulocyte counts on the day of admission than survived healthy dogs in a study of 39 parvovirus-infected puppies.

Castro et al. (2013) investigated the hematobiochemical profile of CPV infected dogs and found significantly higher leukopenia, lymphopenia, and thrombocytopenia as compared to control group.

Schoeman et al. (2013) stated that non-survivors have significantly lower total leucocyte, neutrophil, band neutrophil, lymphocyte and eosinophil count than survivors at admission to hospital. A lack of cytopenia, specifically normal total leucocyte and lymphocyte counts, had a positive predictive value of 100% for survival 24 h post admission. The puppies that were destined for recovery had a rebound increase in the lymphocyte count 24 h after admission.

Shah et al. (2013) observed anaemia (PCV 22.18 ± 1.21), lymphocytopenia (12.23 ± 1.95) and thrombocytopenia (90.09 ± 3.25) in the fifteen CPV positive dogs. Leukocytopenia was observed in 42.8% of cases and thrombocytopenia was observed in 57.1% of cases.

Salem (2014) observed decrease in RBCs count, haemoglobin concentration, PCV percentage, leucocytes count, neutrophil and lymphocyte CPV-infected dogs.

Dongre et al. (2015) stated lower Hb, PCV, and TEC values in CPV-infected dogs, which could be attributed to haemorrhage, blood loss in faeces, and haemodilution caused by intestinal fluid absorption and compensatory reabsorption of water by the kidney, as well as leukopenia with neutrophilia and lymphopenia.

Sulthana (2015) observed reduced mean values of Hb, PCV, TEC and TLC indicating anaemia and leukopenia on haematological analysis in CPV infected dogs.

Parthiban et al. (2016) studied twenty PCR confirmed CPV cases of non-descript pups in the age group of 6-8 weeks and revealed low white blood cells (WBC) count in 80% of pups ($3-5 \times 10^3/\mu\text{L}$), low red blood cells (RBC) count in 60% of pups ($4-5 \times 10^6/\mu\text{L}$), low haemoglobin (Hb) values in 50% of pups (6-11 g/dl) however, the differential leukocyte counts showed neutropenia in 85% of pups and lymphopenia in 60% of pups.

Andrea et al. (2017) recorded anaemia as evidenced by low values of PCV (30.5 ± 2.7), Hb (11.46 ± 0.87) TEC (5 ± 0.41) and MCV (56.22 ± 1.99) along with neutropenia (59.48 ± 1.59) and lymphocytosis (29.13 ± 1.48) in CPV affected dogs.

Bhargavi et al. (2017) recorded prominent haematological changes that includes anaemia, lymphopenia, thrombocytopenia and neutrophilia in CPV infected dogs.

Dash et al. (2017) observed decreased mean haemoglobin, total erythrocyte count, packed cell volume, total leucocyte count with neutropenia and lymphocytosis in 58 cases which were found positive by Kit Ag test method.

Kumar and Kumar (2017) observed that mean haemoglobin concentration, PCV and TEC decreased significantly in CPV infected dogs. There was significant increase in neutrophil and decrease in lymphocyte, whereas non-significant change noticed in eosinophil, monocytes and basophils.

Thakur and Thakur (2018) studied haemato-biochemical alterations in two groups affected with canine parvovirus. Group A with 6 healthy pups and group B with 6 CPV infected pups. They observed significant ($P<0.05$) decrease in Hb, PCV, TEC and MCHC in Group B when compared to Group A, but there was significant ($P<0.05$) increase in MCV in Group B dogs as compared to healthy dogs.

Gaykwad et al. (2018) observed abnormalities in haematological parameters of 18 CPV infected dogs, with significantly higher ($p<.05$) mean Hb concentration and RBC count followed by marked leukopenia and neutropenia ($p<.05$) as compared to control dogs before treatment.

Terzungwe (2018) observed abnormalities in haematology of the 29 dogs with CPVE, in which anaemia was seen in 20.7%, thrombocytopenia 3.5%, pancytopenia {leucopenia 31.0%, neutropenia 55.5% and lymphopenia 3.5%}, leucocytosis 27.6%, neutrophilia 17.2%, eosinophilia 3.5%, lymphocytosis 3.5% and monocytosis 65.5%. He revealed, neutropenia in the acute phase of the disease while monocytosis and lymphocytosis were encountered after the hyperplasia of cell lines of the bone marrow.

Ahmad et al. (2020) reported low white blood cells (WBC) count, low red blood cells (RBC) counts, low haemoglobin (Hb) values and differential leukocytic counts having neutropenia and lymphopenia in canine parvo virus affected dogs.

Alves (2020) revealed mean PCV of 38.3%, TLC of 3,523 cells/ μ L, PLT of 272,000 cells/ μ L. Although mean PCV was within reference interval, 48.5% of dogs in the study had anaemia. Thrombocytopenia was present in 24.8% of CBC's and leukopenia was observed in 85.1% of cases, mainly due to neutropenia and lymphopenia, which were observed in 97.5% and 88.9% of CBC's, respectively.

El-Zahar et al. (2019) recorded that dogs infected with canine parvovirus showed significant decreased haemoglobin, RBCs, PCV% and leukocytes with

lymphopenia and neutropenia compared to healthy control dogs, whereas the platelets, monocytes, eosinophils and basophils did not differ significantly.

Kataria *et al.* (2020) conducted study in 36 dogs from which 21 dogs were found positive for CPV infection. The haematological parameters showed non-significant lower values of haemoglobin and non-significantly higher values of PCV and TLC. Differential leucocyte count revealed neutrophilia, lymphopenia, whereas no changes observed in eosinophils and monocytes.

Khare *et al.* (2020) reported that the mean value of haemoglobin, packed cell volume, TEC, monocyte and lymphocyte decreased significantly while mean value of neutrophil and eosinophil increased in affected dogs.

Mehta *et al.* (2020) recorded decrease haemoglobin concentration on day of presentation in 34 CPV infected dogs, and concluded that the decline in haemoglobin concentration can be attributed to haemorrhagic enteritis owing to damaged capillaries of intestinal villi and haemodilution caused due to persistent fluid therapy.

Harizan *et al.* (2021) observed that, the mean values of Hb, PCV, TEC and TLC were significantly ($p < 0.01$) decreased in dogs affected with parvoviral enteritis as compared to healthy dogs and the DLC showed significant neutrophilia and lymphopenia as compared to healthy dogs.

2.6 Biochemical Analysis

Nappert *et al.* (2002) studied 25 puppies naturally infected with canine parvovirus infection and recorded significantly lower plasma concentration of sodium, potassium, chloride and bicarbonate than healthy dogs in control group.

Yilmaz and Senturk (2007) conducted a study on serum biochemical parameters in dogs infected with parvoviral enteritis and reported elevated serum indicators of tissue injury (ALT, creatinine kinase myocardial isoenzyme [CK-MB], blood urea nitrogen and creatinine).

Sagar *et al.* (2008) reported decrease in mean blood glucose, total serum protein, albumin and globulin whereas mean values of A/G ratio was found to be increased in CPV infected dogs.

Roy *et al.* (2010) reported marked decrease in serum glucose value, serum total protein, globulin whereas non-significant decrease in albumin value in CPV affected groups on day of presentation.

Bhat *et al.* (2012) studied plasma concentration of creatine kinase-MB isoenzyme in dogs with intestinal form of canine parvovirus-2 infection and found 18 positive sample out of 45 faecal samples by PCR. Further, they observed increased levels of CK-MB in CPV infected dogs compared to healthy dogs. These results indicated about fourfold increase in the CK-MB level in the infected dogs.

Bastan *et al.* (2013) reported higher urea and creatinine concentrations in 39 parvovirus-infected puppies than healthy dogs on the day of admission.

Castro *et al.* (2013) observed canine parvovirus infection was correlated with leukopenia, lymphopenia, thrombocytopenia, hypoglycemia, and hypoproteinemia whereas canine corona virus infection was only related with lymphopenia.

Schoeman *et al.* (2013) observed that severe hypokalaemia, hyponatraemia and hypochloraemia, hypoalbuminemia, decrease in plasma proteins and decrease in plasma citrulline concentrations and reduced total blood calcium concentrations, however, high serum C-reactive protein (CRP), the elevated blood urea, creatinine and inorganic phosphate in CPV infected dogs.

Shah *et al.* (2013) observed elevated BUN, creatinine, AST, ALT, ALP in 75% cases, whereas TP and albumin level were decreased in 80% and 66% cases.

Burchell *et al.* (2014) studied the concentration of chloride in 42 CPV-affected dogs and found that 20 dogs had metabolic acidosis, 10 had metabolic alkalosis, and 12 dogs had a neutral impact. They concluded that CPV enteritis

has considerable disturbances in electrolyte and albumin. Chloride is the most relevant variable in the pathophysiology of acid-base disturbances.

Salem (2014) reported reduction in albumin concentration with increased activities of ALT and ALP, bilirubin, triglycerides and BUN along with a decrease in sodium and chloride concentration in CPV-infected dogs.

ER and OK (2015) studied the levels of cardiac biomarkers and coagulation profiles in dogs with parvoviral enteritis in twenty-seven dogs with parvoviral enteritis (experiment group) and 6 healthy dogs (control group) which were aged between 1.5 and 6 months, weighted between 5 - 15 kg, wherein, they observed significant increase in CK-MB ($P < 0.05$) and BNP ($P < 0.001$) levels in parvoviral enteritis affected dogs compared to healthy dogs, however, no significant change was detected in cardiac troponin I level.

Bhargavi *et al.* (2017) noticed significant elevation of blood urea nitrogen (BUN) however, significant reduction of serum potassium level, in CPV infected pups.

Kumar and Kumar (2017) observed significant reduction in total serum protein, albumin, globulin, serum sodium, potassium, chloride and blood glucose levels, however, significant increase in SGOT level were noticed in CPV infected dogs.

Thakur and Thakur (2018) observed significant increase of AST and ALP levels, however significant decreased in albumin level in canine parvovirus infected pups.

Acciaccia *et al.* (2020) recorded reduction in albumin concentration in CPV infected dogs on day of presentation.

Gulersoy *et al.* (2020) observed serum I-FABP and CK-MB levels which were statistically significantly higher in dogs with parvoviral enteritis at 0th hour than in healthy dogs ($p < 0.05$), whereas no statistically significant difference was found at 48th hour between the groups. Serum TFF-3 and cTnI levels were

significantly higher in dogs with parvoviral enteritis at 0th and 48th hour than in the healthy dogs ($p < 0.05$) and no significant difference was found in dogs with parvoviral enteritis at 0th and 48th hour of serum CLDN-3 levels compared to the healthy dogs

Khare *et al.* (2020) reported that TP (g/dl), albumin (g/dl) and globulin (g/dl) levels were significantly lower, while there was high rise in ALT (U/L) and AST (U/L) activity observed in CPV infected dogs.

Kumar *et al.* (2020) recorded significant increase in alkaline phosphatase whereas significant decrease was noticed in total serum protein, albumin, globulin and AG ratio in CPV affected dogs.

Venn *et al.* (2020) studied serum D-lactate concentrations in dogs with parvoviral enteritis which included dogs with CPV undergoing treatment ($n = 40$) and healthy control dogs ($n = 9$), in which they found CPV dogs had a higher D-lactate concentration. While no relationship was identified between serum D-lactate concentrations and markers of CPV disease severity, acid-base status, or outcome.

Harizan *et al.* (2021) observed that, the mean values of serum glucose, serum total protein, albumin and globulin were significantly lower ($P < 0.01$) in CPV affected dogs than healthy dogs. The mean values of serum ALT and AST were significantly ($P < 0.05$ and $P < 0.01$, respectively) higher whereas, no significant difference was found in serum creatinine level in infected dogs as compared to healthy dogs.

2.7 Treatment

Prittie (2004) advised fluid therapy to correct dehydration, reestablishment of circulating blood volume, to correct electrolyte and acid-base disturbances, and restorations of fluid loss through vomition and diarrhoea.

Duffy *et al.* (2010) assessed the effectiveness of rcG-CSF in dogs with parvovirus-induced neutropenia, in which 28 dogs with parvovirus and

neutropenia were treated with rcG-CSF @5 µg/kg of rcG-CSF (Amgen Inc., Thousand Oaks, CA, USA) subcutaneously for every 24 h once daily until the patient's neutrophils resumed reference range, or until death or euthanasia occurred and outcomes were compared to those of 34 dogs with parvovirus. They observed that the treatment with rcG-CSF was effective in stimulating neutrophil recovery and shortening the duration of hospitalization in dogs with parvovirus infection.

Savigny and Macintire (2010) administered Oseltamivir (2 mg/kg, PO, q 12 h) in the treatment of canine parvo viral enteritis along with standard therapy in 35 dogs. Treatment group dogs received oseltamivir, while control group dogs received an equivalent volume of placebo. Dogs in the treatment group had increased survival rate and gained a significant percentage of weight during hospitalization as compared to the control group. Dogs in the treatment group had no significant decline in WBC count for any of the initial 5 days of hospitalization while control group experienced a significant decrease in WBC count from entry Day 1 to Days 2, 3, and 4 of hospitalization. In addition, they stated that oseltamivir use did not associate with any major adverse clinical effects.

Pillai and Deepa (2010) treated CPV positive two months old spitz dog with fluids 30ml DNS and 50ml Ringer's lactate, antibiotic Cefipine at 20mg/kg i/v, Pantoprazole, Stemetil and Ethamsylate; the treatment was continued for 4 days and considerable recovery was noticed by 5th day of treatment.

Nivy et al. (2011) administered I/V fluids at 7.5 ml/kg/h with 5% dextrose supplementation, Ampicillin @ 25 mg/kg, I/V, q 8h, Gentamicin @ 6.6 mg/kg, I/V, q 24h, Maropitant @ 1 mg/kg/SC/q24h, Metoclopramide @ 1.5 mg/kg/d IV at constant infusion rate, Famotidine @ 1 mg/kg, I/V, q 24h, Ivermectin @ 400 µg/kg, S/C and Praziquantel @ 6 mg/kg, S/C to treat 10-week-old dog affected with parvovirus.

Singh et al. (2011) treated 50 CPV infected dogs with symptomatic therapy with Intacef @ 20 mg/Kg body weight intramuscular for five days, Injection Perinorm @ 0.1 mg/Kg body weight intramuscularly for three days,

Injection Stryptochrome @ 0.1-0.2 mg/Kg body weight intramuscular for two days, Injection Tribivet @ 2 ml intramuscularly for five days and Ringer lactate solution @ 5-10 ml/Kg body weight intravenous for three days. Out of these 50 treated dogs, 43 recovered completely within 3-5 days and 4 dogs died after first day treatment and 3 dogs on second day of treatment.

Banja et al. (2002) symptomatically administered intravenous ringer's lactate solution of dextrose normal saline @ 45-90 ml/kg b.wt. metoclopramide @ 1.0 mg/kg b.wt. adrenochrome monosemicarbazone @ 0.1 -0.2 mg/kg b.wt. and gentamycin @ 4.0 mg/kg b.wt. and successfully saved 90.1%, 100% and 80.9% dogs suffering from CPV (Single), CCV (Single) and CPV and CCV (Mixed) infections within five days of therapy respectively.

Bastan et al. (2013) used a standard protocol that included continuous intravenous (I/V) crystalloids (Lactated Ringers) supplemented with potassium chloride, dextrose %, amino acid solution, and metoclopramide @ 0.2 mg/kg I/V for every 6- 8 hour (0.2 mg/kg), and ampicillin @ 15 mg/kg I/V every 8 hours to treat CPV affected puppies successfully.

Bhat et al. (2013) divided CPV-infected dogs into two groups and treated group I with levofloxacin @ 5mg/kg b.wt., I/V, OID for 3 days, ondansetron @ 0.2 mg/kg b.wt., I/M or I/V, BID for 2 days, ethamsylate @ 10mg/kg b.wt., I/V or I/M, BID for 2 days, fluid therapy with RL and DNS and Tribivet @ 1.0 -1.5 ml on alternate days while group II was administered ceftriaxone @ 20mg/kg b.wt., BID I/M or I/V for 3 days and all other drugs remained same as in group I. They concluded that therapy regimen of group I was shown to be more successful than that of group II.

Dongre et al. (2013) treated 8 parvoviral infected dogs with fluids (RL, I/V), antibiotics ciprofloxacin @ 10 mg/kg b.wt., BID, I/V, prochlorperazine @ 0.5 mg/kg b.wt., OID, I/M, ranitidine @ 0.5 mg/kg b.wt., BID, I/M, dicyclomine @ 0.1 mg/kg b.wt., I/M, haemocoagulase @ 0.5-1 ml total dose, TID, I/V and ascorbic acid @ 100 mg/day, I/V and concluded that treatment's efficacy was found to be 62.50 percent.

Albaz et al. (2015) evaluated the Acyclovir as antiviral agent in prophylactic treatment of puppies experimentally infected by canine parvovirus 2 and found that the Acyclovir regime was effective in prevention of CPV2 replication in puppies.

Parthiban et al. (2015) treated 20 affected pups with suspension Cefpodoxime @ 10 mg/kg PO, Inj. Tribivet (Thiamine + Pyridoxine + Cyanocobalmin) @1ml IM and Inj. Intalyte @10 ml/kg IV for 5 days and complete recovery observed within five days in fourteen pups while two pups takes seven days, whereas four pups with severe leucopenia and neutropenia died during course of treatment.

Parthiban et al. (2016) treated 20 pups affected pups with Inj. Cefotaxime sodium @ 20 mg/kg iv. Inj. Perinorm @ 0.2 mg/kg iv, Inj. Pantoprazole 1 mg/kg, iv, Inj. DNS @10 ml/kg iv and Inj. Ringer's lactate @ 10 ml/kg iv BID for 5 days. With aggressive therapy and supportive care 70% of pups were recovered within five days of therapy 15% of pups were recovered after five days of therapy.

Ahmad et al. (2020) treated 5 dogs successfully with Ringer lactate infusion along with Ranitidine @ 2ml/IV, Metoclopramide @ 0.5mg/kg body weight, antibiotic Ceftriaxone Sodium @ 2ml/IV for 5 days, Tranexamic acid 5mg/kg body weight, Ondansetron IV for 3 days. For diarrhoea, Metronidazole drip of 100 mL, and for anaemia due to severe bloody diarrhoea Haemaccel drip 20mL for each puppy used on 3rd to 5th day.

Venn et al. (2020) administered the isotonic crystalloid fluids (120 mL/kg/d IV) with potassium chloride supplementation, maropitant citrate (1 mg/kg IV q24h), cefoxitin (22 mg/kg IV q8h) and dextrose supplementation in inpatient group, while administered 8 mg/kg cefovecin sodium SC once, SC fluids (Normosol-R, 30 mL/kg q6h), maropitant citrate (1 mg/kg SC q24h), as well as PO potassium (Tumil-K) and dextrose (Karo syrup) supplementation to outpatient group. While dogs in both groups were syringe fed Hill's a/d (1 mL/kg q6h) and provided buprenorphine (0.02 mg/kg IV or SC) as needed for abdominal

discomfort and Ondansetron (0.5 mg/kg IV or SC) was given to the dogs with vomiting that was refractory to maropitant.

Suvethika and Kumar (2021) advised symptomatic and supportive treatment for parvo infected dogs which includes 5% Ringer Lactate, Hydroxyethyl starch infusion along with antibiotic Ceftriaxone Sodium, Pantaprazole, Metronidazole and Ondansetron intravenously along with tender coconut water and electoral water orally however they advised Tranexamic acid as haemostatic agent to control bleeding.

Muñoz *et al.* (2021) studied immunomodulator (human Dialyzable Leukocyte Extract-hDLE) as a therapeutic aid in puppies affected by Canine Parvovirus type II and found significant increment of the survival rate in puppies co-administered with the immunomodulator. Further they concluded that the immunomodulator are responsible for the augment of puppies' survival.

MATERIALS AND METHODS

The present study entitled "**THERAPEUTIC MANAGEMENT OF PARVO VIRUS INFECTION IN DOGS**" was carried out in the Department of Veterinary Clinical Medicine, Ethics & Jurisprudence, Nagpur Veterinary College, Nagpur. Clinical cases suspected of Canine Parvovirus presented at Veterinary Clinical Complex (VCC), Nagpur Veterinary College, Nagpur, were chosen for the present study.

3.1 Selection of Animals

In the present study, dogs of age groups ranging from zero to twelve months, different breeds, and gender presented at Veterinary Clinical Complex, Nagpur Veterinary College, Nagpur and showing clinical signs suggestive of Parvovirus infection were included. A thorough anamnesis, clinical examination, haematological and serum biochemical tests examination were undertaken.

Dogs with the chief complaint of anorexia, foul-smelling diarrhoea, vomition, dehydration, depression were considered for screening.

3.2 Screening

The fecal samples were collected from the rectum of suspected animals by using a sterile swab. The primary screening of suspected cases was performed by using Anigen Rapid CPV Ag Test Kit (BioNote, Inc. 22 Samsung 1-ro 4-gil, Hwaseong-si, Gyeonggi-do 18449, Republic of Korea) a rapid immunochromatography-based test. The test device has a testing window. The testing window has an invisible T (test) zone and C (control) zone. When a sample is applied into the S (sample) hole on the device, the sample will laterally flow on the surface of the test strip. If there is enough parvovirus antigen in the sample, a visible T band will appear. The C band should always appear after a sample is applied, indicating a valid result. By this means, the device can indicate the presence of canine parvovirus antigen in the sample.

3.3 Rapid Antigen Test Kit

3.3.1 Kit composition

Each test kit contains the following (Plate 3.1)

- One test device
- One disposable dropper
- One bottle containing assay buffer 2 ml
- One sterile swab for sample collection

3.3.2 Test Procedure

Canine faeces were collected with a swab by directly inserting into the rectum (Plate 3.2), and the swab was placed into a sample tube containing 2 ml assay buffer. The swab in buffer mixed well (Plate 3.3), until sample has been dissolved properly and from the mixed sample add 4 drops into sample hole (S) (Plate 3.4), drop by drop vertically. The test results were read at 10 min.

3.3.3 Interpretation of results

- **POSITIVE:** Two distinct coloured lines appear. One line should be in the control line region (C) and another line should be in the test line region (T).
- **NEGATIVE:** One coloured line appears in the control line region (C). No apparent coloured line appears in the test line region (T).
- **INVALID:** Control line (C) fails to appear. Insufficient specimen volume or incorrect procedural techniques are the most likely reasons for control line failure.

The dogs showing positive for CPV Rapid Test kit were considered as affected cases of CPV infection and subjected to therapeutic assessment. Another fecal sample was taken for detection of canine parvovirus (CPV) infection using the PCR technique.

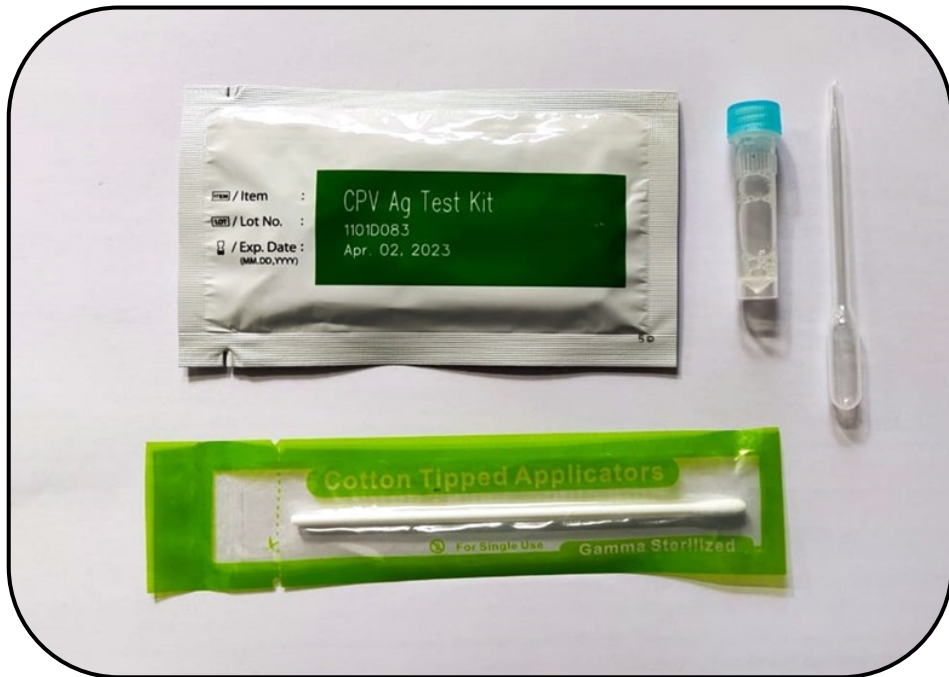


Plate 3.1: Rapid Antigen Test Kit



Plate 3.2: Collection of faecal sample by using sterile swab



Plate 3.3: Mixing of swab in buffer



Plate 3.4: Adding of mixed sample into sample hole

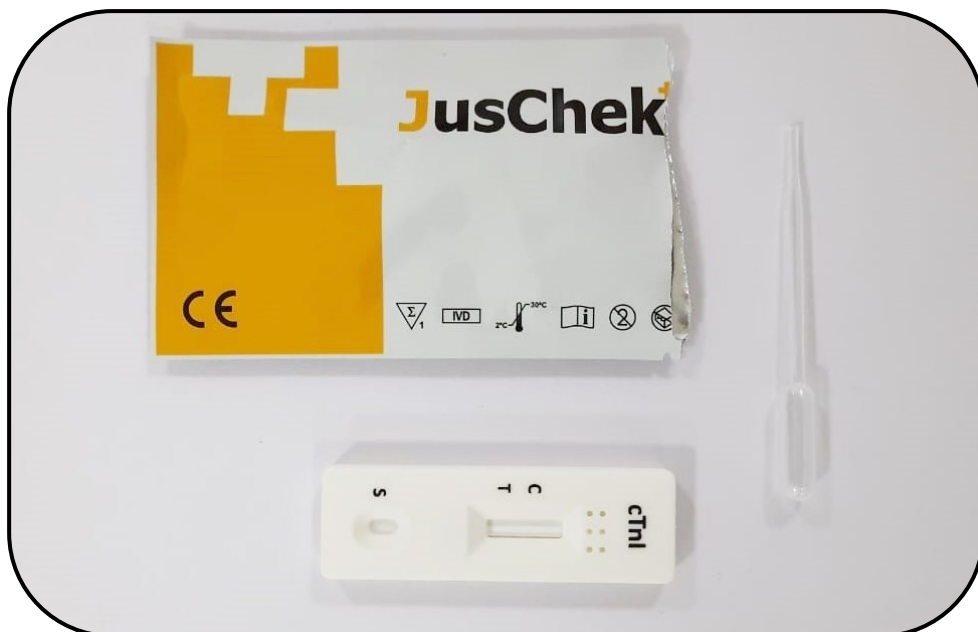


Plate 3.5: Cardiac Troponin I Rapid Test Kit

3.4 Clinical observations:

General clinical examination of dogs comprising of patient's age, sex, breed, rectal temperature, respiration rate, cardiac rate, conjunctival examination, frequency of diarrhea/ dysentery, consistency, colour of faeces, and vomitus, if any, was carried out. The degree of dehydration was also assessed by skin tent test.

3.5 Laboratory material

3.5.1 Chemicals and reagents

The laboratory kits used for estimating various biochemical constituents namely TP, serum albumin, and CK-MB, manufactured by Coral Clinical Systems a Division of Tulip Diagnostics (P) Ltd. Pantnagar, Uttarakhand - 263 153, India.

3.5.2 Glass wares and plastic wares

Glass vials of 5 ml and 10 ml capacity, test tubes and beakers were cleaned, washed and sterilized by hot air oven and stored till use. Single use sterile blood collection vials were used. Pipettes and syringes (10 and 20 ml) were also used in the present study.

3.6 Collection of clinical samples

3.6.1 Whole Blood

Blood samples of dogs were collected by venipuncture from the cephalic or saphenous vein in clean and sterile syringes and transferred into Ethylene-Diamine Tetra-Acetate (EDTA) vials were gently mixed for CBC analysis.

3.6.2 Serum

Blood samples were collected in the clot activator vial for serum isolation. The clot activator vial was left stable for 1 hour and then centrifuged for serum

separation. Serum was collected in a dry eppendorf tube by a micropipette with care, from which biochemical parameters were estimated.

3.6.3 Faeces

The fecal samples of suspected dogs were collected aseptically with a sterile swab. Collected samples were used to detect canine parvovirus with a CPV Rapid Test kit manufactured by BIONOTE and Polymerase Chain Reaction technique. Fecal samples were stored at -20°C by adding 10% Phosphate Buffer Saline solution (PBS) for detection of CPV virus using Polymerase Chain Reaction.

3.7 Blood/ Plasma Examination:

3.7.1 Haematological Analysis

Hematological estimations Hb, PCV, TEC's, TLC, and DLC, were carried out using Hematology Analyser (Horiba Make ABX Micros ESV-60) on the principle of photometry numeric integration and electronic impedance variation methods.

3.7.2 Biochemical Analysis

3.7.2.1 Estimation of Serum Total Protein (gm/dL)

The serum total protein was estimated using the Biuret endpoint method using Microlab 300 Biochemical Semi-Auto Analyser.

3.7.2.2 Estimation of Serum Albumin (gm/dL)

Serum Albumin was estimated by BCG endpoint method using Microlab 300 Biochemical Semi-Auto Analyser.

3.7.2.3 Estimation of Serum Creatine Phosphokinase-MB (IU/L)

Levels of plasma CK-MB activity were determined by immune-inhibition methodology using a CK-MB kit. The values were expressed as IU/L.

3.7.2.4 Cardiac Troponin I Rapid Test

The cardiac troponin I (cTnI) test were carried out by Cardiac Troponin I Rapid Test Kit (Plate 3.5) (Whole Blood/Serum/Plasma) is a rapid chromatographic immunoassay for the qualitative detection of cardiac Troponin I in whole blood, serum or plasma as an aid in the diagnosis of myocardial infarction (MI).

3.7.2.4.1 Principle

The cTnI Test Device (Whole Blood/Serum/Plasma) is a qualitative, membrane based immunoassay for the detection of cTnI in whole blood, serum or plasma. The membrane is pre-coated with capture reagent on the test line region of the test. During testing, the whole blood, serum or plasma specimen reacts with the particle coated with anti-cTnI antibodies. The mixture migrates upward on the membrane chromatographically by capillary action to react with capture reagent on the membrane and generate a coloured line. The presence of this coloured line in the test line region indicates a positive result, while its absence indicates a negative result. To serve as a procedural control, a coloured line will always appear in the control line region indicating that proper volume of specimen has been added and membrane wicking has occurred.

3.7.2.4.2 Materials Provided

- Test devices (contain anti-cTnI antibody coated particles and capture reagent coated on the membrane)
- Buffer
- Droppers
- Package insert

3.7.2.4.3 PROCEDURE

Allow test device, specimen, and/or controls to reach room temperature (15- 30°C) prior to testing.

1. Bring the pouch to room temperature before opening it. Remove the test device from the sealed pouch and use it as soon as possible.
2. Place the test device on a clean and level surface.
3. For Serum or Plasma specimens: Hold the dropper vertically and transfer 2 drops of serum or plasma (approximately 100 μL) to the specimen well (S) of the test device, and start the timer.
4. For Venipuncture Whole Blood specimens: Hold the dropper vertically and transfer 1 drop of venipuncture whole blood (approximately 50 μL) to the specimen well (S) of the test device, then add 1 drop of buffer (approximately 50 μL) and start the timer.
5. For Fingerstick Whole Blood specimens: To use hanging drops: Allow 1 hanging drops of fingerstick whole blood specimen (approximately 50 μL) to fall into the centre of the specimen well (S) on the test device, then add 1 drop of buffer (approximately 50 μL) and start the timer.
6. Wait for the coloured line(s) to appear. Read results at 10 minutes. Do not interpret results after 20 minutes.

3.7.2.4.4 Interpretation

- **POSITIVE:** Two distinct coloured lines appear. One line should be in the control line region (C) and another line should be in the test line region (T).
- **NEGATIVE:** One coloured line appears in the control line region (C). No apparent coloured line appears in the test line region (T).
- **INVALID:** Control line (C) fails to appear. Insufficient specimen volume or incorrect procedural techniques are the most likely reasons for control line failure.

3.8 Polymerase Chain Reaction for CPV

3.8.1 Viral DNA extraction

3.8.1.1 Introduction

The genomic DNA of CPV-2 from the stool samples was extracted using QIAamp® Fast DNA Stool Mini Kit. The QIAamp Fast DNA Stool Mini Kit quickly and easily purifies total DNA from fresh or frozen stool samples. The purified DNA is of high quality and well suited for use in PCR and other downstream applications. Purification requires no phenol-chloroform extraction or alcohol precipitation and involves minimal handling. DNA is eluted in low-salt buffer and is free of protein, nucleases, and other impurities or inhibitors. The purified DNA is ready for use in PCR and other enzymatic reactions or can be stored at -30°C to -15°C for later use. Stool samples typically contain many compounds that can degrade DNA and inhibit downstream enzymatic reactions. To ensure the removal of these substances, the QIAamp Fast DNA Stool Mini Kit contains InhibitEX Buffer, which is specially formulated to separate inhibitory substances from DNA in stool samples.

3.8.1.2 Principle

The QIAamp Fast DNA Stool Mini Kit is designed to purify total DNA from up to 220 mg stool rapidly and is suitable for fresh and frozen samples. A special protocol is provided for isolating DNA from larger amounts of stool. The fast and easy procedure comprises the following steps:

- Lysis of and separation of impurities from stool samples in InhibitEX Buffer
- Purification of DNA on QIAamp Mini spin columns

3.8.1.3 Procedure

The following steps were used to isolate DNA from stool samples.

- Weigh 180–220 mg stool in a 2 ml microcentrifuge tube (not provided) and place tube on ice.

- Add 1 ml InhibitEX Buffer to each stool sample. Vortex continuously for 1 min or until the stool sample is thoroughly homogenized.
- Centrifuge sample for 1 min to pellet stool particles.
- Pipet 25µl Proteinase K into a new 1.5ml or 2ml microcentrifuge tube
- Pipet 600 µl supernatant from step 3 into the 1.5 ml or 2 ml microcentrifuge tube containing Proteinase K.
- Add 600µl Buffer AL and vortex for 15s.
- Incubate at 70°C for 10 min.
- Add 600µl of ethanol (96–100%) to the lysate, and mix by vortexing.
- Carefully apply 600 µl lysate from step 8 to the QIAamp spin column. Close the cap and centrifuge for 1 min. Place the QIAamp spin column in a new 2 ml collection tube, and discard the tube containing the filtrate. Repeat step 9 until all lysate is loaded.
- Carefully open the QIAamp spin column and add 500 µl Buffer AW1. Centrifuge for 1 min. Place the QIAamp spin column in a new 2 ml collection tube, and discard the collection tube containing the filtrate.
- Carefully open the QIAamp spin column and add 500 µl Buffer AW2. Centrifuge for 3 min. Discard the collection tube containing the filtrate.
- Place the QIAamp spin column in a new 2 ml collection tube and discard the old collection tube with the filtrate. Centrifuge for 3 min.
- Transfer the QIAamp spin column into a new, labeled 1.5 ml microcentrifuge tube and pipet 50 µl Buffer ATE directly onto the QIAamp membrane. Incubate for 1 min at room temperature, then centrifuge for 1 min to elute DNA. If yield will be quantified by UV absorbance, blank the measuring device using Buffer ATE to avoid false results.

- Transferred the elute to a fresh capped 2 ml collection tube for longer DNA

Table 3.1 QIAamp Fast DNA Stool Mini Kit Contents

Number of preps 50	50
QIAamp Mini spin columns	50 ml
Collection Tubes (2 ml)	200 ml
InhibitEX [®] Buffer	140 ml
Buffer AL* 33	33 ml
Buffer AW1* (concentrate) 19	19 ml
Buffer AW2 [†] (concentrate) 13	13 ml
Buffer ATE 12	12 ml
Proteinase K 1.4	1.4 ml
Quick-Start Protocol	1

* Contains chaotropic salt. Take appropriate laboratory safety measures and wear gloves when handling. Not compatible with disinfecting agents containing bleach. See page 6 for safety information.

† Contains sodium azide as a preservative.

3.8.2 Primers for PCR

Two sets of primer pairs, Pab, which detects CPV types 2ab and 2b designed for this study, described by (Sheikh et al. 2017). Primer pair CPV-2ab-F sense (5'-GAAGAGTGGTTGTAAATAATT -3') and CPV-2ab-R sense (5'-CCTATATAACCAAAGTTAGTAC -3') located respectively, at 3025 - 3045 and 3685-3706 of the CPV genome, yields a 681 base pair (bp) product.

3.8.3 PCR reaction

The PCR was performed in a thermal cycler, each consisting of initial denaturation at 95 °C for 5 min followed by 30 cycles of 95 °C for 1 min, annealing at 50°C for 30 sec and extension at 72 °C for 1 min and a final extension at 72 °C for 10 min. PCR products were electrophoresed on 1% agarose

gels, gels were stained with Ethidium bromide and then visualized under UV light and documented by gel documentation system.

Table 3.2 Composition of the reaction mixture for PCR

Component	Quantity (µl)
PCR Master mix	12.5
Nuclease free water	7.5
Forward primer	1
Reverse primer	1
DNA Template	2.5
Total	24.5

3.9 Therapeutic Management

A total of 48 cases found positive for canine parvovirus infection after screening with Rapid CPV Ag Test Kit were included for various therapeutic trials. The dogs were divided into four groups based on age into 0-3 months, 4-6 months, 7-9 months, and 10-12 months old and were placed randomly in 2 different therapeutic groups, i.e., T1 and T2 comprising of 24 animals in each group with following treatment regimens:

- Group I comprised of the use of antibiotics with supportive and symptomatic therapy.
- Group II comprised the use of antiviral (Oseltamivir), antibiotic, symptomatic and supportive therapy.

The therapeutic management, Different drugs used as per the protocol and their Doses, Route, presented in Table 3.3 and Table 3.4.

Table 3.3 Grouping of dogs

Groups	Treatment	
	T1	T2
Group 1 0 to 3 months	<ul style="list-style-type: none"> • Inj. Ringer's Lactate and D.N.S. • Inj. Ceftriaxone+Tazobactam @20 mg/Kg I/V BW, bid, 	<ul style="list-style-type: none"> • Inj. Ringer's Lactate and D.N.S, • Inj. Ceftriaxone+Tazobactam @20 mg/Kg I/V BW, bid,
Group 2 4 to 6 months	<ul style="list-style-type: none"> • Inj. Ondansetron @0.5 mg/Kg BW, bid, I/M • Inj. Pantoprazole @1 mg/Kg BW, od, I/V 	<ul style="list-style-type: none"> • Inj. Ondansetron @0.5 mg/Kg BW, bid, I/M • Inj. Pantoprazole @1 mg/Kg BW, od, I/V
Group 3 7 to 9 months	<ul style="list-style-type: none"> • Inj. Ethamsylate @250-500 mg, bid, I/V or I/M, 	<ul style="list-style-type: none"> • Inj. Ethamsylate @250-500 mg, bid, I/V or I/M,
Group 4 10 to 12 months	<ul style="list-style-type: none"> • Inj. Amino acid preparation (if required) 	<ul style="list-style-type: none"> • Inj. Amino acid preparation (if required) • Oseltamivir @2 mg/Kg BW, bid, Oral.

The evaluation of therapeutic regimens was done based on changes in clinico-hematological parameters on day 0 (before treatment) and on 5th day after treatment.

3.10 Statistical Analysis

The collected data was statistically analyzed following the methods of Snedecor and Cochran (2004).

RESULTS AND DISCUSSION

Canine parvovirus infection is one of the highly contagious and most fatal disease in dogs. The treatment strategies implemented to canine parvoviral enteritis is mainly the symptomatic one, though intermittent attempts have been made by researchers and practicing veterinarians to introduce different therapeutic agents to treat Canine parvovirus infection. Likewise, the efforts were made by using antiviral drug as one of the therapeutic management under the present study entitled **“THERAPEUTIC MANAGEMENT OF PARVO VIRUS INFECTION IN DOGS”**. The study was carried out in the Department of Veterinary Clinical Medicine, Ethics and Jurisprudence, Veterinary Clinical Complex (VCC) of Nagpur Veterinary College, Nagpur. The objectives of the study were to record the prevalence of canine parvovirus infection in dogs and to study the diagnosis and comparative therapeutic efficacy of different drugs in canine parvovirus infection in dogs.

The footfall of dog population presented to Veterinary Clinical Complex, Nagpur Veterinary College from October 2020 to September 2021 were screened for Canine parvovirus infection. Dogs showing chief complaint of anorexia, foul-smelling diarrhoea, vomition, dehydration, depression and suspected for canine parvovirus infection were further subjected to clinical examination, screening by using Antigen Rapid CPV Ag Test Kit, and haemato-biochemical examination. The prevalence of canine parvovirus enteritis was studied for a period between October 2020 to September 2021. Out of these, forty-eight dogs randomly selected were divided into four groups based on age into 0-3 months, 4-6 months, 7-9 months and 10-12 months old and were subjected to 2 different therapeutic protocols, i.e., T1 and T2 comprising of 24 animals in each group. Therapeutic management was carried out with conventional therapy and antiviral drug therapy consisting of ‘Oseltamivir’.

4.1 Diagnostic Results

4.1.1 Detection of canine parvoviral antigen in faecal samples

4.1.1.1 Rapid CPV Ag Test Kit

Faecal samples of dogs suspected for CPV were tested with immunochromatography based kit, of which 48 dogs found positive for the infection were selected for the study. Positive and negative results with BioNote Rapid CPV Ag Test Kit are depicted in Plate 4.1 and 4.2.

4.1.1.2 Polymerase Chain Reaction

DNA was isolated from the faecal samples and subjected to PCR for further confirmation. A total of 48 samples were tested by PCR and all were found positive evident by an amplicon of 681 bp on 1% agarose gel as depicted in Plate 4.3.

4.2 Prevalence

Prevalence studies can be used to establish a baseline for future investigations and diagnosis. These studies not only help veterinary practitioners to diagnose the disease, but also help the general population to become more aware of warning indications.

A total of 8,970 canine patients were presented to VCC, Nagpur Veterinary College, Nagpur from October 2020 to September 2021, out of which total 173 dogs were diagnosed with canine parvovirus infection indicating a total of 1.93 percent affection.

4.2.1 Age-wise Prevalence

The age-wise prevalence of Canine parvovirus infection was collected and presented in Table 4.1 and graphically depicted in Fig. 4.1.

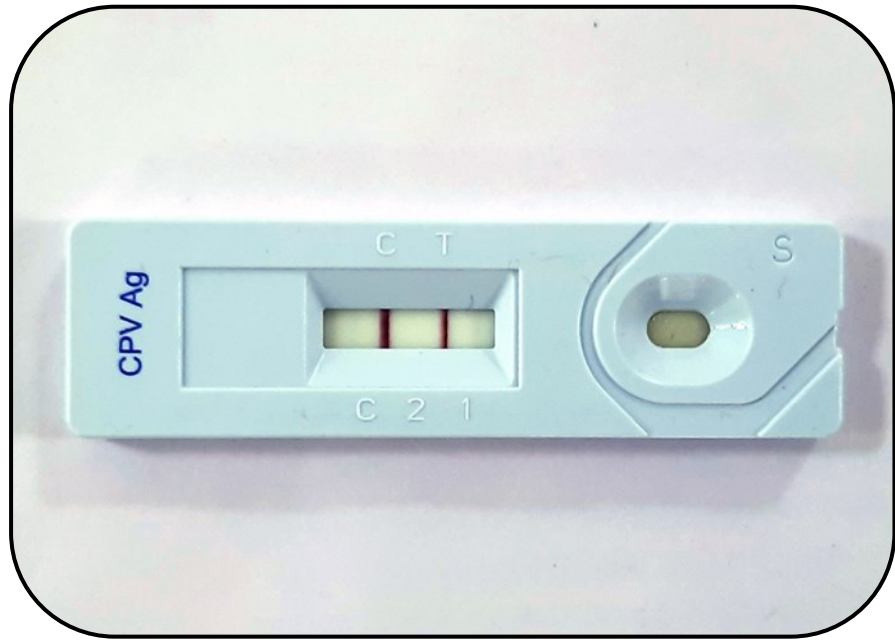


Plate 4.1: Rapid Antigen Test Kit showing positive result

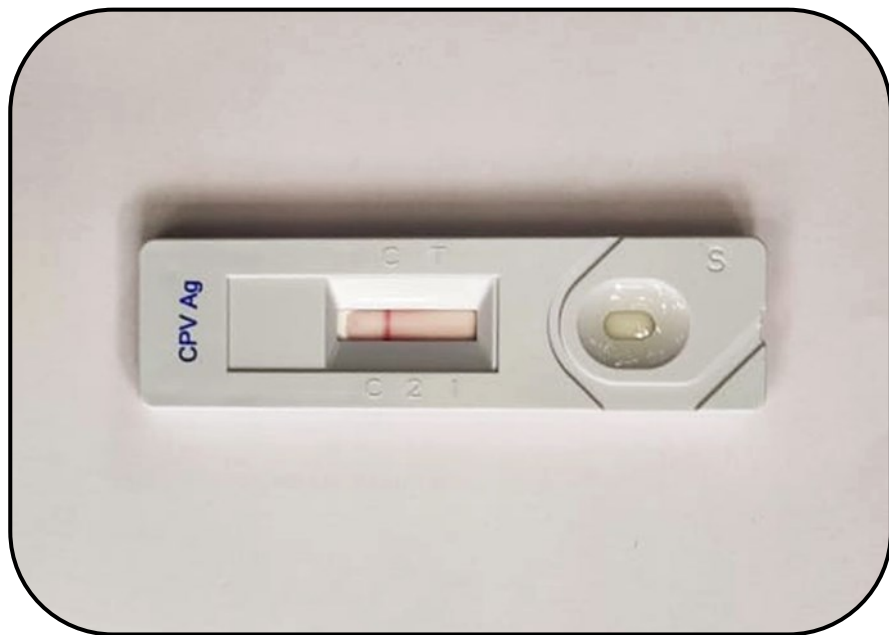


Plate 4.2: Rapid Antigen Test Kit showing negative result

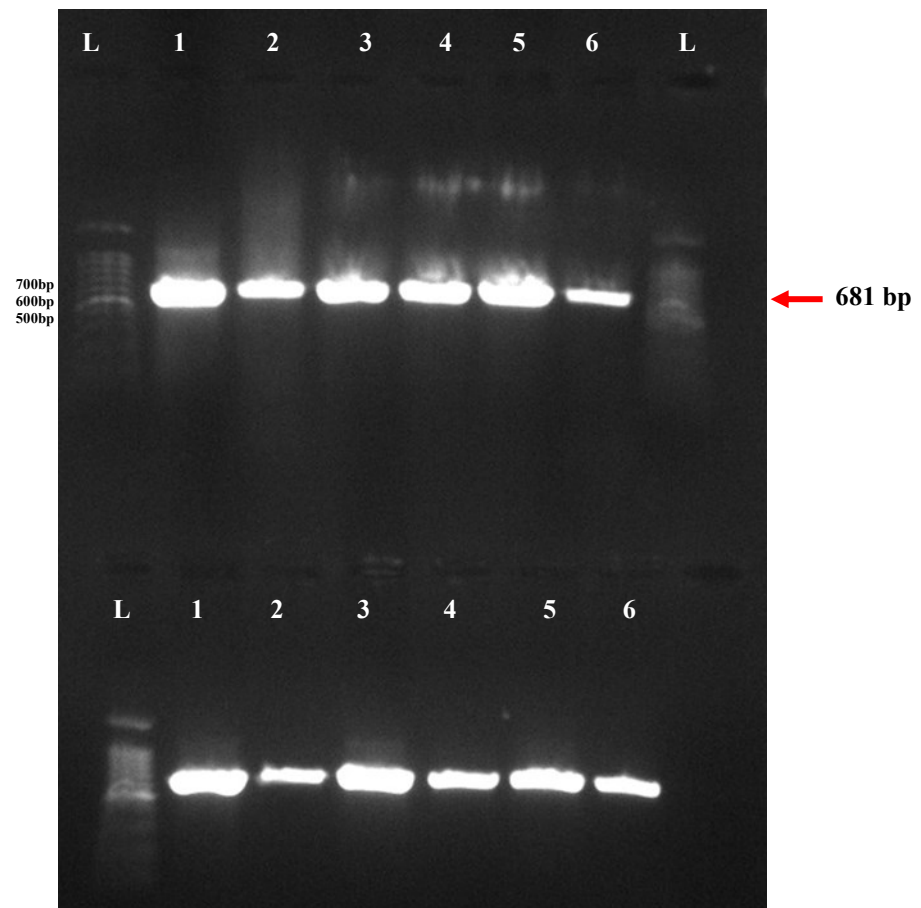


Plate 4.3: Agarose Gel Electrophoresis: 1% agarose gel electrophoresis with ethidium bromide revealed amplification of 681 bp as reported by Sheikh *et al.*,2017

Table 4.1 Age-wise prevalence of dogs affected with Canine parvovirus infection

Age	No. of dogs affected	Percentage of dogs affected (%)
0-3 months	73	42.20
4 to 6 months	53	30.63
7 to 9 months	22	12.72
10 to 12 months	17	9.83
>12 months	8	4.62
Total	173	100

As per the data represented in Table 4.1, it was observed that the highest affection of Canine parvovirus enteritis was found in the age group of 0–3 months with 42.20 percent, followed by dogs of age between 4 to 6 months (30.63%). However, 12.72 percent of dogs suffering from canine parvovirus infection belong to 7 to 9 months of age and 9.83 percent of dogs belong to 10-12 months of age, respectively. The lowest prevalence was found in dogs with age more than 1 year (4.62%). Thus, it was concluded that young dogs, mostly less than of 6 months age are more vulnerable to canine parvovirus infection, on the other side adult dogs, more than a year age group were least affected.

In the present study, the dogs between 0-3 months of age showed highest prevalence which is in agreement with Khare *et al.* (2019) and Reddy *et al.* (2015), however the lowest prevalence (9.83%) was reported in 9-12 months which is similar to that of Archana *et al.* (2009). Even though the average age group susceptible for CPV infection was 5-7 months (Alves, 2020 and Singh *et al.* 2013). The young animals <6 month of age were found to be more susceptible for the infection which could be due to the virus's preference for fast proliferating intestinal crypt cells in weaning pups with a higher mitotic index as a result of changes in bacterial flora, lack of protective immunity due to lack of maternal antibody, variant strain and probably had not been vaccinated or incomplete vaccination status of pups. On the contrary, the lower prevalence rate of CPV was reported in dogs >6 month of age might be due to acquired immunity because of constant exposure to field viruses.

4.2.2 Sex-wise Prevalence

The data regarding sex-wise prevalence of Canine parvovirus infection is presented in Table 4.2 and depicted in Fig 4.2.

Table 4.2 Sex-wise prevalence of dogs affected with Canine parvovirus infection

Sex	No. of dogs affected	Percentage of dogs affected (%)
Male	101	58.38
Female	72	41.62
Total	173	100

The data indicated that the occurrence of canine parvovirus infection was higher in males (58.38%) than females (41.62%) with respect to total number of dogs with Canine parvovirus enteritis. The findings of this study are in agreement with Parthiban *et al.* (2010), Reddy *et al.* (2015), Khare *et al.* (2019), and Sharma *et al.* (2019). The significant difference in sex wise prevalence was not recorded by Archana *et al.* (2009), but non-significantly higher prevalence in male was noted by Nahat *et al.* (2015) than female, however, significantly higher prevalence in female than male was reported by Chethan *et al.* (2021).

In the present study, male dogs were found to be more susceptible for the infection which could be due to higher footprints of males than female in VCC. The more chances of exposure of males to CPV infection observed because of their behaviour and the pet owners' selected preference for keeping males as pets. It is also speculated that males usually move more than females, hence makes them more susceptible to field infections.

4.2.3 Breed-wise prevalence

The data regarding the breed-wise prevalence of Canine parvovirus infection cases was collected and presented in Table 4.3 and graphically depicted in Fig. 4.3

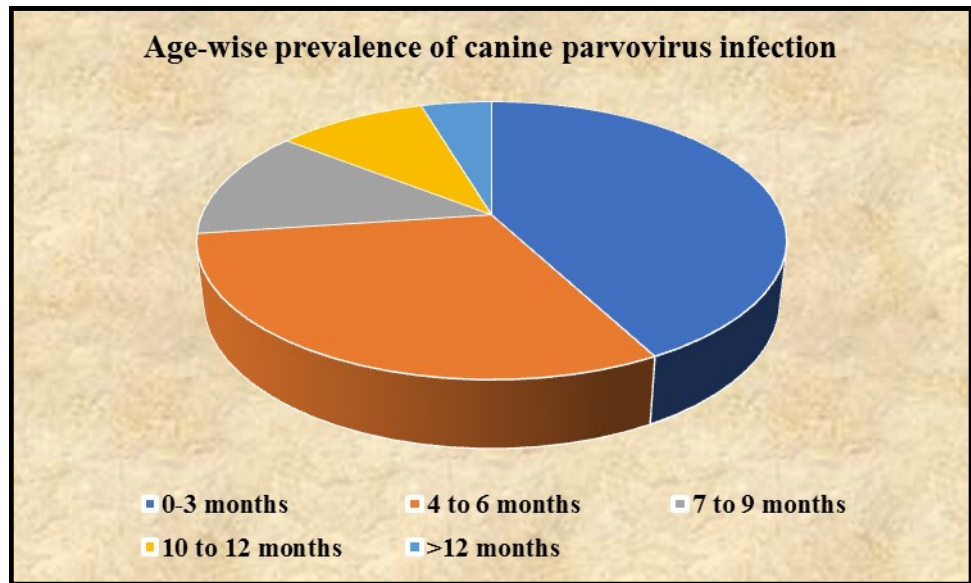


Fig. 4.1: Age-wise prevalence of canine parvovirus infection

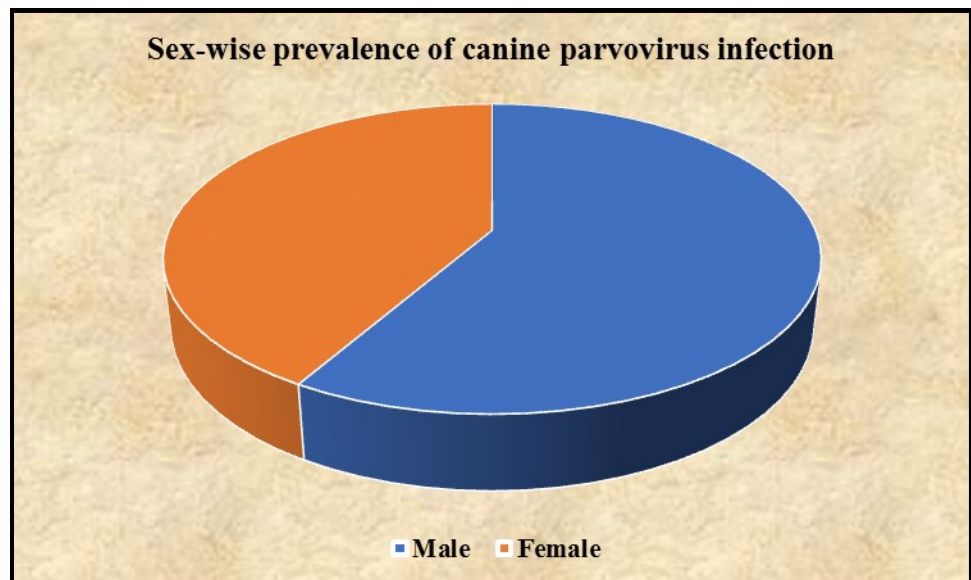


Fig. 4.2: Sex-wise prevalence of canine parvovirus infection

Table 4.3 Breed-wise prevalence of dogs affected with canine parvovirus infection

Breed	No. of dogs affected	Percentage of dogs affected (%)
Non-descript	61	35.26
Labrador	43	24.85
Doberman	18	10.40
German Shepherd	15	8.67
Rottweiler	9	5.20
Spitz	7	4.05
Golden Retriever	5	2.89
Lhasa Apso	4	2.31
Pug	3	1.73
Great Dane	3	1.73
St. Bernard	3	1.73
Boxer	2	1.15
Total	173	100.00

In this investigation, canine parvovirus infection was found to be most prevalent in non-descript dogs (35.26%), followed by Labradors (24.85%), Doberman (10.40%), German Shepherd (8.67%), Rottweiler (5.20%), Spitz (4.05%) Golden Retriever (2.89%), Lhasa Apso (2.31%), however Pug, Great Dane, St. Bernard breeds showed an equal prevalence of 1.73%. The lowest prevalence of 1.15% was recorded in Boxer breed.

The present study revealed highest prevalence in non-descript dogs (35.26%), which is in corroboration with Archana *et al.* (2009), Hasan *et al.*, (2017), Khare *et al.* (2019), whereas, Sharma *et al.* (2019), Tanwar *et al.* (2020), and Chethan *et al.* (2021) documented that CPV infection was more prevalent in Labrador Retriever. On the contrary Kumar *et al.* (2010), Singh *et al.* (2013) and Reddy *et al.* (2015) reported highest prevalence of CPV in German Shepherds, Doberman and Spitz, respectively. In current study non-descript breed shows highest prevalence of CPV infection which might be attributed to a lack of vaccination in these breeds and the fact that the majority of them were stray so

they have a habit of roaming around and even entering hospital premises, where they can easily pick up an infection. Labrador dogs are at second position in prevalence which could be because of, the fact that it is most reared breed in this region, and its sociable breed trait combined with easy maintenance which makes this breed very appealing to pet parents.

4.2.4. Month wise Prevalence

The data regarding the month-wise prevalence of Canine parvovirus infection cases was collected and presented in Table 4.4 and graphically depicted in Fig. 4.4

Table 4.4 Months-wise prevalence of dogs affected with Canine parvovirus infection

Months	No. of dogs affected	Percentage of dogs affected (%)
October	5	2.89
November	20	11.56
December	9	5.20
January	10	5.78
February	8	4.62
March	7	4.05
April	12	6.94
May	5	2.89
June	7	4.05
July	40	23.12
August	29	16.76
September	21	12.14
Total	173	100.00

In this investigation, Canine parvovirus infection was found to be most prevalent in month of July (23.12%), followed by August (16.76%), September (12.14%) November (11.56%), January (5.78%), April (6.94%), December

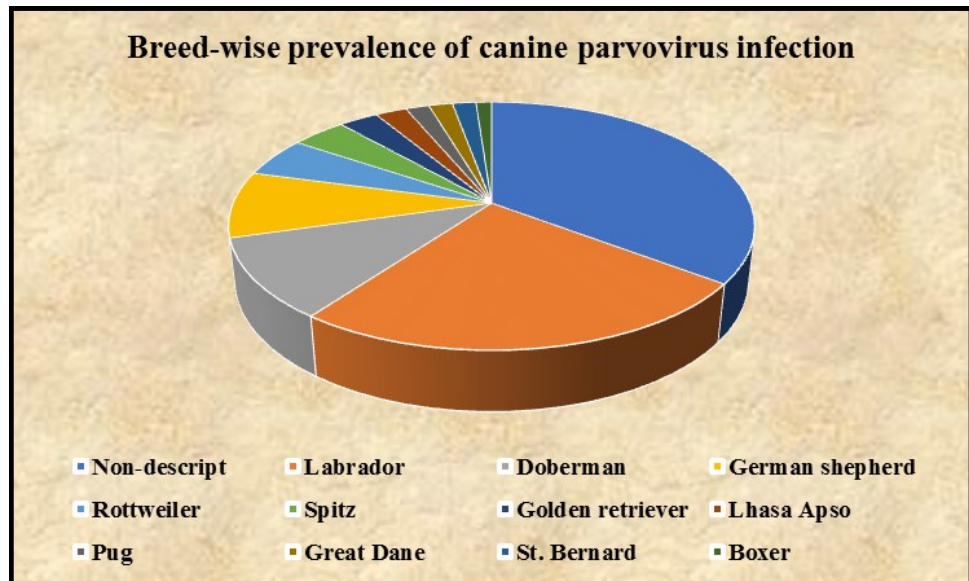


Fig. 4.3: Breed-wise prevalence of canine parvovirus infection

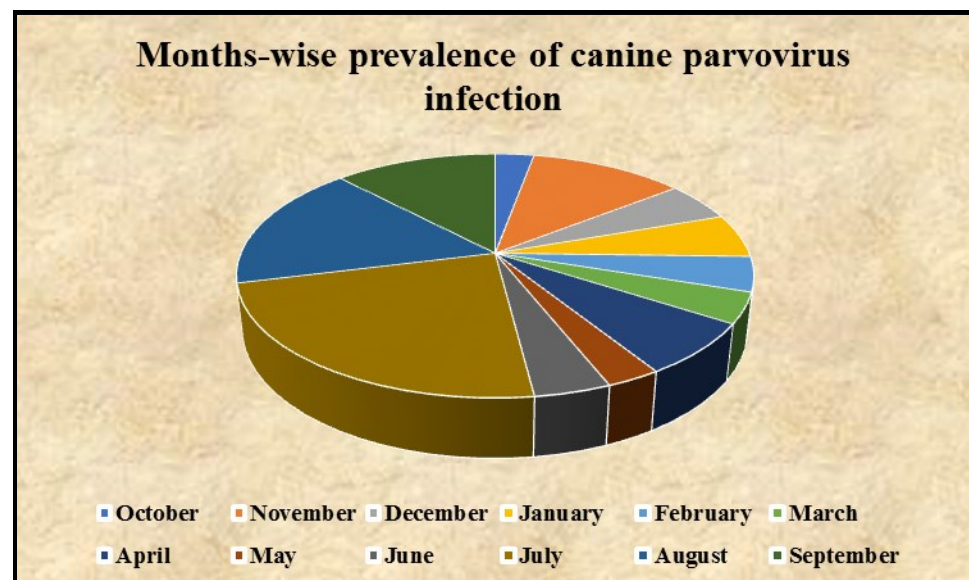


Fig. 4.4: Months-wise prevalence of canine parvovirus infection

(5.20%), February (4.62%), March (4.05%) and June (4.05%), however May and October showed an equal and lowest prevalence of 2.89%.

Canine parvovirus infection was found most prevalent in month of July (23.12%), which is in agreement with the Reddy *et al.* (2015), whereas Mehta *et al.* (2017) reported highest prevalence (65.00%) in the month of February although Sharma *et al.* (2019) found highest prevalence of CPV infection in spring season followed by summer and winter season.

The highest prevalence was reported in July which could be because, CPV is normally dead or latent over the winter, therefore most cases observed in seasons other than winter.

4.3 Physical examination

4.3.1 Rectal temperature

The mean \pm SE values of rectal temperature ($^{\circ}$ F) of 48 dogs diagnosed with Canine parvovirus infection-was 102.08 ± 0.23 $^{\circ}$ F. However, 9 (18.75%) dogs had pyrexia, 33 (68.75%) dogs had normal rectal temperature and the rest 6 (12.5%) had subnormal body temperature. The increased body temperature may be suggestive of viremia in early stage of disease, while late reported case had subnormal body temperature due to severe fluid and electrolyte losses (Banja 2002, and Kumar 2017).

4.3.2 Heart Rate

The mean \pm SE values of heart rate of 48 dogs diagnosed with Canine parvovirus infection was 143.44 ± 3.39 per min. Tachycardia and hypotension may be attributed to decreased tissue perfusion generated by dehydration, which is secondary to gastrointestinal loss of fluid and proteins (Prittie 2004 and Alves 2020).

4.3.3 Respiration Rate

The mean \pm SE values of respiration rate of 48 dogs diagnosed with Canine parvovirus infection-was 41.70 \pm 1.96 per min. Tachypnoea may be a consequence of the response to abdominal pain, frequent in dogs with parvovirus infection, or a compensatory response to a possible metabolic acidosis (Prittie 2004 and Alves 2020).

4.3.4 Colour of mucous membrane

Out of 48 dogs diagnosed with Canine parvovirus infection in the present study, 62.5% of dogs were found to normal mucous membrane, pale mucous membrane in 20.83% of dogs (Plate 4.4) and remaining 16.67% of dogs showed congested mucosa (Plate 4.5).

Normal mucus membrane appears pink and moist. Pale mucus membrane may be due to anaemia, decreased tissue perfusion or both. Anaemia is common haematological finding in CPV enteritis, especially in the later phases of disease. This causes due to suppression of erythropoiesis, as circulating red blood cells have a long half-life relative to the short period during which the virus suppresses production in the bone marrow (Goddard *et al.* 2008). Also due to the loss of blood due to gastric and intestinal haemorrhages (Bhargavi *et al.* 2017).

4.3.5 Colour and consistency of faeces

In the present study out of 48 cases, haemorrhagic diarrhoea was observed in 40 (83.33%) dogs (Plate 4.6) and non-haemorrhagic diarrhoea was observed in 8 (16.67%) dogs. Considering the consistency of faeces out of 48 dogs, 45 (93.75%) dogs had watery faeces and remaining 3 (6.25%) had soft faeces on presentation. The current observation and interpretation are based on the CPV virus's natural tendency to destroy the "villi" and "microvilli" structure by dividing at Crypts of Lieberkuhn, compromising their ability to replace cells at the villi in order to maintain an adequate assimilatory barrier and surface area. Bloody diarrhoea and haemorrhagic enteritis are caused by a vascular barrier impairment



Plate 4.4: Pale mucous membrane

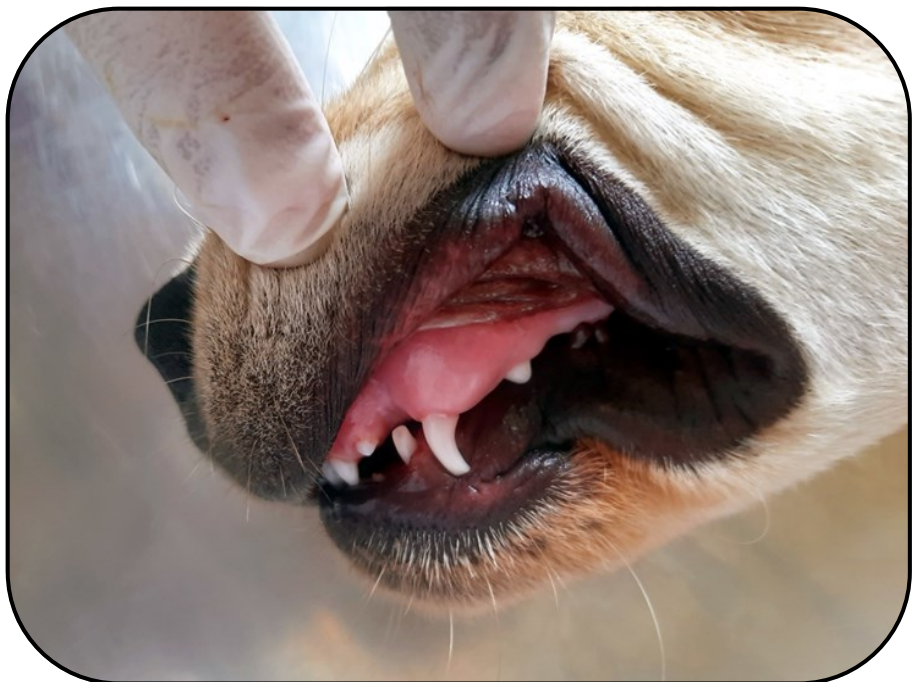


Plate 4.5: Congested mucous membrane



Plate 4.6: Haemorrhagic diarrhoea of watery consistency



Plate 4.7: Whitish and frothy vomitus of CPV infected dog

and continual cellular sloughing of villi (Rabbani *et al.* 2021), which is suggestive of regurgitation of haemorrhagic duodenal contents (Kumar and Kumar, 2017).

4.3.6 Colour and consistency of vomition

In the present study, out of 48 cases, all the dogs had episodes of vomiting. Out of 48 dogs, whitish vomiting of frothy nature was observed in 11 (22.92%) dogs (Plate 4.7), plain watery type of vomiting was observed in 30 (62.5%) dogs, haematemesis was observed in 6 (12.5%) dogs and only one dog (2.08%) had yellow colour of vomiting.

4.4 Clinical signs

Dogs affected with Canine parvovirus infection showed various clinical signs which includes depression, lethargy, anorexia, fever, vomiting, haemorrhagic diarrhoea, dehydration.

The clinical signs exhibited by 48 dogs was depression to lethargy (Plate 4.8), followed by inappetence (100%), pale mucosa (20.83%), fever (25%), vomiting (100%), haemorrhagic diarrhoea (83.33%), non-haemorrhagic diarrhoea (16.67), and dehydration (83.33%) (Plate 4.9). Similar clinical signs were recorded by Banja *et al.* (2002), Goddard *et al.* (2008), Sagar *et al.* (2008), Singh *et al.* (2011), Decaro and Buonavoglia (2012), Bastan *et al.* (2013), Castro *et al.* (2013), Salem (2014) and Gulersoy *et al.* (2020).

The data regarding the clinical signs showed by dogs with Canine parvovirus infection was collected and presented in Table 4.5 and graphically depicted in Fig. 4.5.

4.5 Vaccination status

In the present study, out of 48 cases, 33 (68.75%) dogs were non-vaccinated for parvovirus vaccine, 12 (25%) dogs were fully vaccinated and 3 (6.25%) dogs had taken only first dose of vaccine.

Table 4.5: Clinical signs observed in dogs with canine parvovirus infection

Clinical signs	No. of dogs affected	Percentage of dogs affected (%)
Depression to lethargy	48	100
Inappetence	48	100
Pale mucosa	10	20.83
Fever	12	25
Vomiting	48	100
Haemorrhagic Diarrhoea	40	83.33
Non-haemorrhagic Diarrhoea	8	16.67
Dehydration	40	83.33

The present study revealed highest prevalence of CPV in non-vaccinated dogs, which is in accordance with Parthiban *et al.* (2010), Hasan *et al.*, (2017), Alves (2020), and Mehta *et al.* (2017). Though Sharma *et al.* (2019) reported high prevalence of canine parvo infection in non-vaccinated dogs (42.70%), but no disease prevalence was recorded in vaccinated dogs.

The higher occurrence of CPV infection in non-vaccinated dogs could be due to a lack of protective immunity, whereas the lower occurrence of CPV infection in vaccinated dogs suggested that existing vaccinations provided enough protection.

4.6 Haemato-biochemical parameters

Haematological parameters provide data on how the body is functioning and also it may indicate abnormalities long before clinical indications are recognized. During the current study, several haematological parameters were examined at regular intervals between the groups. The results for various haemato-biochemical parameters were analyzed statistically by applying Three-Way ANOVA using Factorial Completely Randomized Design. The results are presented below.

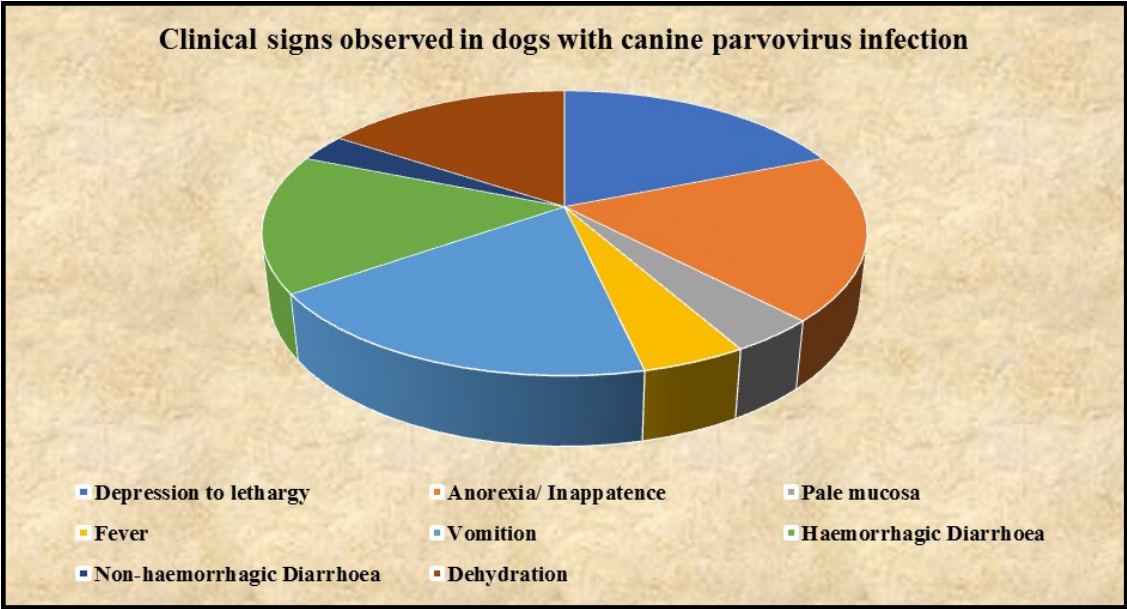


Fig. 4.5: Clinical signs observed in dogs with canine parvovirus infection



Plate 4.8: Depressed and lethargic dog



Plate 4.9: Dog showing severe dehydration

4.6.1 Haemoglobin

The mean haemoglobin (g/dL) value in both treatment groups at scheduled intervals is presented in Table 4.6 and graphically depicted in Fig 4.6.

Table 4.6 Mean Haemoglobin (g/dL) of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	12.56±0.56 ^{Ba}	9.86±0.97 ^{Aa}	11.21±0.6 ^a
	4-6	13.65±0.67 ^{Ba}	12.33±0.76 ^{Ab}	12.99±0.52 ^b
	7-9	13.70±1.2 ^{Ba}	10.83±0.75 ^{Aa}	12.26±0.80 ^{ab}
	10-12	15.71±0.86 ^{Bb}	13.36±1.37 ^{Ab}	14.53±0.85 ^c
	Pooled mean for age, T1	13.90±0.46 ^B	11.59±0.54 ^A	12.75±0.39
T2	0-3	12.41±0.73 ^{Ba}	10.41±0.33 ^{Aa}	11.41±0.49 ^a
	4-6	14.86±1.04 ^{Bb}	12.31±0.66 ^{Ab}	13.59±0.70 ^b
	7-9	14.80±1.07 ^{Bb}	11.01±0.54 ^{Aab}	12.90±0.80 ^{ab}
	10-12	15.85±0.50 ^{Bb}	11.98±0.34 ^{Ab}	13.91±0.65 ^b
	Pooled mean for age, T2	14.48±0.48 ^B	11.43±0.27 ^A	12.95±0.35

Critical Difference (C.D.): For Group: 1.08, For Age: 1.52, For Interval: 1.08

Common capital alphabets within the respective row and common small alphabets within the respective column indicate non-significant difference

It is more vital to maintain haemoglobin levels in order to ensure tissue oxygenation. All of the dogs in this study were tested for haemoglobin, PCV, and TEC to confirm anemia, as this plays a crucial function in transporting oxygen to all the organs.

Table 4.7 Analysis of variance for Haemoglobin (g/dL) levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	55.934	11.187	-
Factor A	1	1.009	1.009	0.251
Factor B	3	108.299	36.100	8.993
Factor C	1	172.378	172.378	42.942
A x B	3	6.181	2.060	0.513
A x C	1	3.286	3.286	0.818
B x C	3	7.613	2.538	0.632
A x B x C	3	4.412	1.471	0.366
Error	75	301.065	4.014	-
Total	95	660.174	-	-

The mean haemoglobin varied significantly ($P < 0.01$) between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 12.56 ± 0.56 , 13.65 ± 0.67 , 13.70 ± 1.21 , and 15.71 ± 0.86 , respectively and the corresponding values on 5th day of treatment were 9.86 ± 0.97 , 12.33 ± 0.76 , 10.83 ± 0.75 , and 13.36 ± 1.37 , respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 12.41 ± 0.73 , 14.86 ± 1.04 , 14.80 ± 1.07 , and 15.85 ± 0.50 , respectively, and the corresponding values on the 5th day of treatment were 10.41 ± 0.33 , 12.31 ± 0.66 , 11.01 ± 0.54 , and 11.98 ± 0.34 , respectively.

In T1 the pooled mean of haemoglobin was observed to be 13.90 ± 0.46 on 0th day which was significantly ($P < 0.01$) higher than 11.59 ± 0.54 , observed on 5th day. In T2 the pooled mean of haemoglobin was observed to be 14.48 ± 0.48 on 0th day which was found to be significantly ($P < 0.01$) higher than 11.43 ± 0.27 , observed on 5th day. The haemoglobin values on 0th day were found to be non-significant in both groups, similarly, on the 5th day, haemoglobin values were found to be non-significant in both the groups. In T1 on the 0th day, the haemoglobin values of the 10-12 month age group were observed to be significantly ($P < 0.01$) higher than those seen in 0-3, 4-6, and 7-9 months age

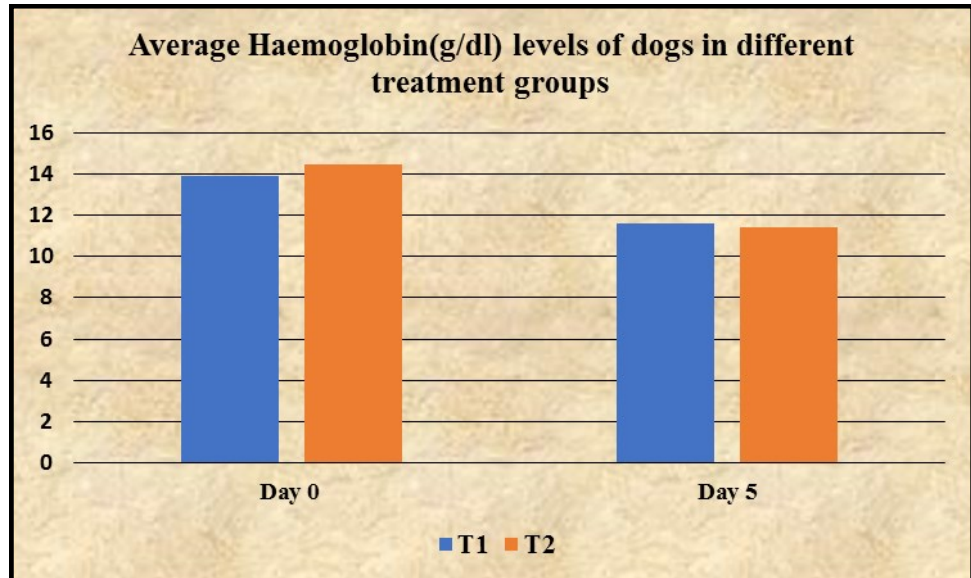


Fig. 4.6: Graphical representation of Average Haemoglobin levels of dogs in different treatment groups

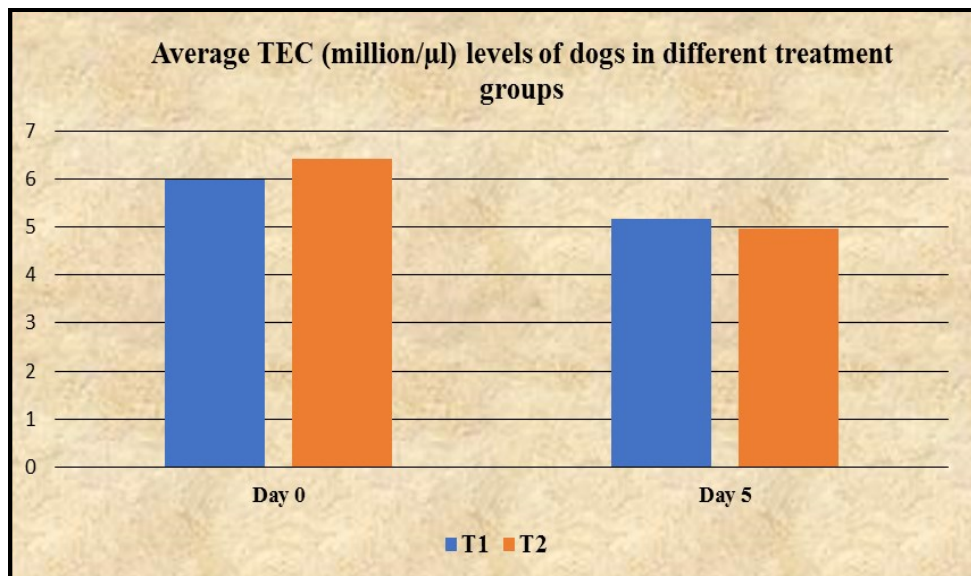


Fig. 4.7: Graphical representation of average TEC levels of dogs in different treatment groups

group. On 5th day, haemoglobin values of 0-3 and 7-9 months age groups were significantly ($P<0.01$) lower than those seen in 4-6 and 10-12 months age groups. In T2 on 0th day, the mean haemoglobin values reported in 0-3 months were significantly ($P<0.01$) lower than that of 4-6, 7-9, and 10-12 months age group. On 5th day, the mean haemoglobin values of 0-3 and 7-9 months age groups were significantly ($P<0.01$) lower than those seen in 4-6 and 10-12 months age groups.

The findings of present study are in agreement with Parthibhan *et al.* (2015), Gaykwad *et al.* (2016) and Terzungwe (2018). The mean haemoglobin concentration observed on 0th day in the present study might be due to excessive fluid loss resulting in dehydration leading to haemoconcentration during parvoviral diarrhoea (Billett 1990, Gaykwad *et al.* 2016 and Kataria *et al.* 2020). Overall, significant decrease in the haemoglobin values was seen from 0th day to 5th day in both the treatment groups than can be attributed to cytotoxic effect of parvo virus on hematopoietic cells and bone marrow which resulted into decreased erythropoiesis (Sharma *et al.* 2008, Goddard *et al.* 2010 and Gaykwad *et al.* 2016), also decline in haemoglobin concentration can be result of haemorrhagic enteritis owing to damaged capillaries of intestinal villi and haemodilution caused due to persistent fluid therapy (Billett 1990 and Mehta *et al.* 2020).

4.6.2 Total erythrocyte count (million/ μ l)

The mean Total erythrocyte count (million/ μ l) value in both treatment groups at scheduled intervals is tabulated in Table 4.8 and graphically depicted in Fig 4.7.

The mean TEC values varied significantly ($P<0.01$) between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 5.59 ± 0.2 , 5.69 ± 0.25 , 6.14 ± 0.60 , and 6.49 ± 0.28 , respectively and the corresponding values on 5th day of treatment were 4.55 ± 0.44 , 5.32 ± 0.32 , 5.07 ± 0.47 , and 5.71 ± 0.55 , respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 5.31 ± 0.35 , 6.68 ± 0.33 , 6.57 ± 0.49 ,

and 7.11 ± 0.39 , respectively, and the corresponding values on the 5th day of treatment were 4.39 ± 0.20 , 5.44 ± 0.32 , 4.67 ± 0.11 , and 5.30 ± 0.17 , respectively.

Table 4.8 Mean Total erythrocyte count (million/ μ l) of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	5.59 ± 0.2 ^{Ba}	4.55 ± 0.44 ^{Aa}	5.07 ± 0.29 ^a
	4-6	5.69 ± 0.25 ^{Ba}	5.32 ± 0.32 ^{Ab}	5.51 ± 0.20 ^{ab}
	7-9	6.14 ± 0.60 ^{Bab}	5.07 ± 0.47 ^{Aab}	5.60 ± 0.39 ^{ab}
	10-12	6.49 ± 0.28 ^{Bb}	5.71 ± 0.55 ^{Ab}	6.10 ± 0.32 ^b
	Pooled mean for age, T1	5.98 ± 0.19 ^B	5.16 ± 0.23 ^A	5.57 ± 0.16
T2	0-3	5.31 ± 0.35 ^{Ba}	4.39 ± 0.20 ^{Aa}	4.85 ± 0.23 ^a
	4-6	6.68 ± 0.33 ^{Bb}	5.44 ± 0.32 ^{Ab}	6.06 ± 0.29 ^b
	7-9	6.57 ± 0.49 ^{Bb}	4.67 ± 0.11 ^{Aab}	5.62 ± 0.37 ^b
	10-12	7.11 ± 0.39 ^{Bb}	5.30 ± 0.17 ^{Ab}	6.20 ± 0.34 ^b
	Pooled mean for age, T2	6.42 ± 0.23 ^B	4.95 ± 0.13 ^A	5.69 ± 0.17

Critical Difference (C.D.): For Group: 0.46, For Age: 0.65, For Interval: 0.46

Common capital alphabets within the respective row and common small alphabets within the respective column indicate non-significant difference

Table 4.9 Analysis of variance for Total erythrocyte count (million/ μ l) levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	11.895	2.379	-
Factor A	1	0.319	0.319	0.427
Factor B	3	17.874	5.958	7.994
Factor C	1	31.270	31.270	41.956
A x B	3	1.870	0.623	0.836
A x C	1	2.545	2.545	3.414
B x C	3	1.704	0.568	0.762
A x B x C	3	1.248	0.416	0.558
Error	75	55.899	0.745	-
Total	95	124.624	-	-

In T1, the pooled mean of TEC was observed to be 5.98 ± 0.19 on 0th day which was significantly ($P < 0.01$) higher than 5.16 ± 0.23 , observed on 5th day. In T2, the pooled mean of TEC was observed to be 6.42 ± 0.23 on 0th day which was found to be significantly ($P < 0.01$) higher than 4.95 ± 0.13 , observed on 5th day. The TEC values on 0th day were found to be non-significantly different in both the groups, similarly, on 5th day, TEC values were found to be non-significantly different in both the groups. In T1 on the 0th day, the TEC values of 0-3 and 4-6 months age group were recorded to be significantly ($P < 0.01$) lower than those seen in 7-9 and 10-12 months age groups. On 5th day, TEC values of 0-3 months age group were observed to be significantly ($P < 0.01$) lower than those seen in 4-6, 7-9, and 10-12 months age groups. In T2 on the 0th day, the mean TEC values were seen in 0-3 months age group were significantly ($P < 0.01$) lower than that of 4-6, 7-9, and 10-12 months age group. On 5th day, the mean TEC values of 0-3 and 7-9 months age groups and were significantly ($P < 0.01$) lower than those seen in 4-6 and 10-12 months age groups.

The findings of the present study are in agreement with Bastan *et al.* (2013), Castro *et al.* (2013), Khare *et al.* (2020), and Harizan *et al.* (2021) who recorded mean TEC values as 5.71 ± 0.42 , 5.7 ± 3.5 , 5.52 ± 0.17 and 6.04 ± 0.23 , respectively on the day of presentation. Terzungwe (2018) observed a mean TEC concentration of 5.82 ± 0.29 in puppies <6 months old and 7.99 ± 0.37 in dogs >6 months of age with CPV infection on the day of presentation. On the whole, a significant decrease in the TEC values was observed from day 0 to day 5 in both the treatment groups.

In the present study, the mean TEC observed on 0th day might be due to excessive fluid loss due to vomiting, diarrhoea, diminished water intake resulting in dehydration leading to haemoconcentration (Weiss & Wardrop 2011 and Gaykwad *et al.* 2016) whereas the decrease in TEC from 0th day to 5th day can be attributed to the cytotoxic effect of parvovirus on hematopoietic cells and bone marrow which results in decreased erythropoiesis (Sharma *et al.* 2008 and Goddard *et al.* 2010) and also damages the capillaries of the intestinal villi leading to loss of blood resulted to reduced total erythrocyte levels.

4.6.3 Packed cell volume (%)

The mean Packed cell volume (%) value in both treatment groups at scheduled intervals is depicted in Table 4.10 and graphically presented in Fig 4.8.

Table 4.10 Mean Packed cell volume (%) of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	36.43±1.90 ^{Ba}	28.83±2.60 ^{Aa}	32.63±1.91 ^a
	4-6	38.98±1.97 ^{Aab}	36.58±2.26 ^{Ab}	37.78±1.47 ^b
	7-9	41.48±3.76 ^{Bb}	33.06±2.39 ^{Aab}	37.27±2.47 ^b
	10-12	49.28±3.72 ^{Bc}	41.63±3.83 ^{Ac}	45.45±2.79 ^c
	Pooled mean for age, T1	41.54±1.71 ^B	35.02±1.64 ^A	38.28±1.26
T2	0-3	36.60±2.5 ^{Ba}	32.05±1.21 ^{Aa}	34.32±1.49 ^a
	4-6	43.76±2.99 ^{Bb}	36.15±1.90 ^{Aa}	39.95±2.04 ^b
	7-9	44.16±3.11 ^{Bb}	33.35±1.53 ^{Aa}	38.75±2.32 ^b
	10-12	48.15±1.64 ^{Bb}	36.03±1.11 ^{Aa}	42.09±2.05 ^b
	Pooled mean for age, T2	43.17±1.50 ^B	34.39±0.77 ^A	38.78±1.05

Critical Difference (C.D.): For Group: NS, For Age: 4.51, For Interval: 3.19

Common capital alphabets within the respective row and common small alphabets within the respective column indicate non-significant difference

The drop in PCV levels causes reduced tissue perfusion, which can lead to ischemic damage. The mean PCV varied significantly ($P < 0.01$) between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 36.43±1.90, 38.98±1.97, 41.48±3.76, and 49.28±3.72, respectively and the corresponding values on 5th day of treatment were 28.83±2.60, 36.58±2.26, 33.06±2.39, and 41.63±3.83, respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 36.60±2.5, 43.76±2.99, 44.16±3.11, and 48.15±1.64, respectively, and the corresponding values on the 5th day of treatment were 32.05±1.21, 36.15±1.90, 33.35±1.53, and 36.03±1.11, respectively.

Table 4.11 Analysis of variance for Packed cell volume (%) levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	504.422	100.884	-
Factor A	1	5.900	5.900	0.168
Factor B	3	1281.612	427.204	12.196
Factor C	1	1403.010	1403.010	40.055
A x B	3	120.862	40.287	1.150
A x C	1	30.600	30.600	0.874
B x C	3	109.887	36.629	1.046
A x B x C	3	62.740	20.913	0.597
Error	75	2627.045	35.027	-
Total	95	6146.080	-	-

In T1 the pooled mean of PCV was observed to be 41.54 ± 1.71 on 0th day which was significantly ($P < 0.01$) higher than 35.02 ± 1.64 , observed on 5th day. In T2 the pooled mean of PCV was observed to be 43.17 ± 1.50 on 0th day which was found significantly ($P < 0.01$) higher than 34.39 ± 0.77 , observed on 5th day. The PCV values on 0th day were found to be non-significantly different in both the groups, similarly, on 5th day, PCV values were found to be non-significantly different in both the groups. In T1 on the 0th day, the PCV values of 0-3n and 4-6 months age group were observed significantly ($P < 0.01$) lower than those recorded in 7-9 months age group which was seen significantly lower than 10-12 month age group. On 5th day, PCV values of 0-3 and 7-9 months age groups were seen to be significantly ($P < 0.01$) lower than the 4-6 months age group which was further significantly ($P < 0.01$) lower than 10-12 months age group. In T2 on 0th day, the mean PCV values seen in 0-3 months were significantly ($P < 0.01$) lower than that of 4-6, 7-9, and 10-12 months age groups. On the 5th day, the mean PCV values of 0-3, 4-6, 7-9, and 10-12 months age groups were non-significant.

The finding of the present study is in compliance with Nappert *et al.* (2002) who recorded mean PCV values as 41.27 ± 10 in dogs. Terzungwe (2018) observed a mean PCV value of 36.45 ± 1.25 in puppies <6 months old and

44.80±1.67 in dogs >6 months of age with CPV infection on the day of presentation. Overall, significant improvement in the PCV values toward normal level was observed from 0th day to 5th day in both the treatment groups.

In the present study, the PCV values toward the higher side on the day of presentation might be due to the effect of vomiting and diarrhoea as well as anorexia and reduced water intake which leads to dehydration which ultimately results in haemoconcentration as noticed by Kataria *et al.* (2020) and Mehta *et al.* (2020). Post-treatment on the 5th day PCV values decreased towards the normal level which might be due to effective rehydration therapy given to dogs to correct dehydration.

4.6.4 Total leucocyte count (10³/µl)

The mean Total leucocyte count (10³/µl) value in both treatment groups at scheduled intervals is presented in Table 4.12 and graphically depicted in Fig 4.9.

Table 4.12 Mean Total leucocyte count (10³/µl) of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	4.51±1.16 ^A	8.80±0.79 ^B	6.66±0.93
	4-6	6.13±1.65 ^A	12.60±3.15 ^B	9.36±1.95
	7-9	5.13±0.93 ^A	10.42±1.41 ^B	7.77±1.13
	10-12	6.18±1.73 ^A	9.45±0.84 ^B	7.82±1.04
	Pooled mean for age, T1	5.49±0.67 ^A	10.31±0.90 ^B	7.90±0.65
T2	0-3	6.77±1.8 ^A	18.26±2.3 ^B	12.52±2.23
	4-6	11.88±4.15 ^A	13.91±2.06 ^B	12.90±2.23
	7-9	4.28±0.67 ^A	15.75±1.23 ^B	10.01±1.85
	10-12	4.73±0.97 ^A	15.38±0.82 ^B	10.05±1.71
	Pooled mean for age, T2	6.92±1.26 ^A	15.82±0.87 ^B	11.37±0.99 [*]

Critical Difference (C.D.): For Group: 2.30, For Age: NS, For Interval: 2.30

Common capital alphabets within the respective row and common small alphabets within the respective column indicate non-significant difference

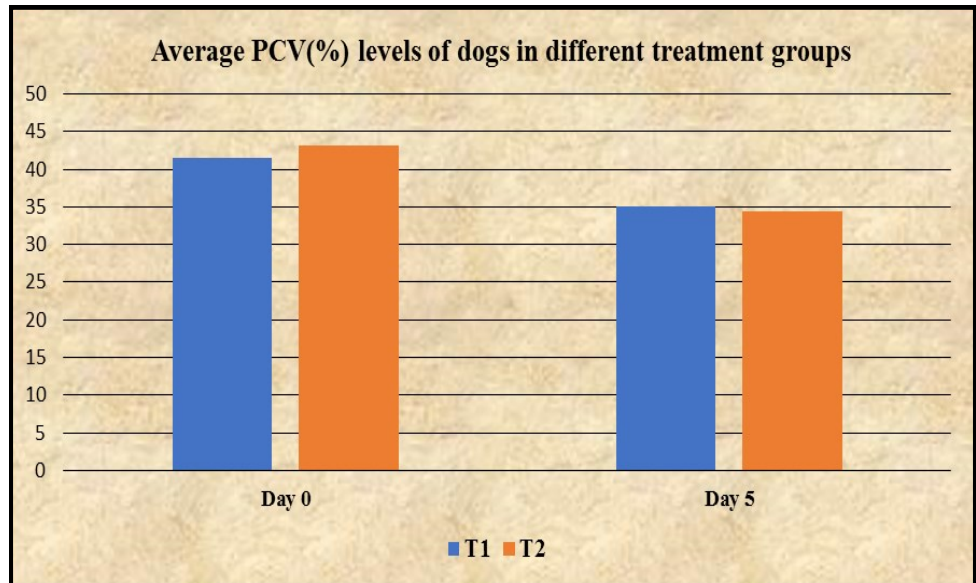


Fig. 4.8: Graphical representation of average PCV levels of dogs in different treatment groups

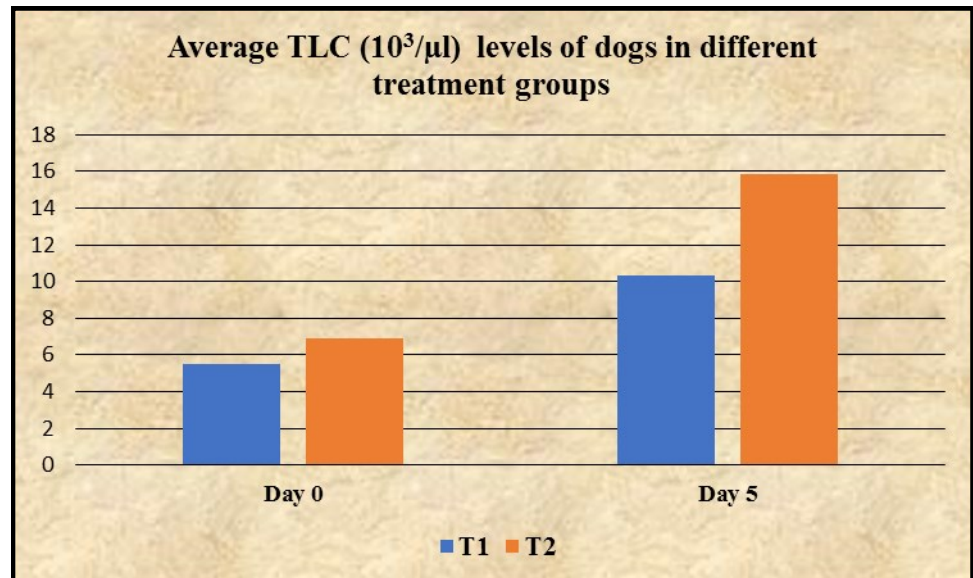


Fig. 4.9: Graphical representation of Average TLC levels of dogs in different treatment groups

Table 4.13 Analysis of variance for Total leucocyte count ($10^3/\mu\text{l}$) levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	287.889	57.578	-
Factor A	1	288.773	288.773	15.801
Factor B	3	78.722	26.241	1.436
Factor C	1	1132.313	1132.313	61.956
A x B	3	52.354	17.451	0.955
A x C	1	100.246	100.246	5.485
B x C	3	61.086	20.362	1.114
A x B x C	3	146.477	48.826	2.672
Error	75	1370.706	18.276	-
Total	95	3518.567	-	-

White blood cells (WBCs) also called leucocytes are immune system cells. They help the body by fighting against infection and thus protect the body. The mean TLC varied non-significantly between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 4.51 ± 1.16 , 6.13 ± 1.65 , 5.13 ± 0.93 , and 6.18 ± 1.73 , respectively and the corresponding values on 5th day of treatment were 6.77 ± 1.8 , 11.88 ± 4.15 , 4.28 ± 0.67 , and 4.73 ± 0.97 , respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 6.77 ± 1.8 , 11.88 ± 4.15 , 4.28 ± 0.67 , and 4.73 ± 0.97 , respectively, and the corresponding values on the 5th day of treatment were 18.26 ± 2.3 , 13.91 ± 2.06 , 15.75 ± 1.23 , and 15.38 ± 0.82 , respectively.

The significant ($P < 0.01$) differences were recorded during the periodic interval for TLC amongst all the age groups for both treatments. The TLC values tend to be increased due to treatment in each age group, indicating the effectiveness of both the treatments at any age of infection. However, the TLC count was increased more significantly ($P < 0.01$) on the 5th day of T2 than T1 indicate the effectiveness of oseltamivir in improving TLC count.

The findings of the present study are in agreement with, Castro *et al.* (2013), Dongre *et al.* (2015), El-Zahar *et al.* (2020), and Mehta *et al.* (2020) who recorded mean TLC of 7.8 ± 3.5 , 5.06 ± 0.12 , 5.61 ± 0.4 and 5.27 ± 0.9 respectively. However, Sulthana (2015) reported a mean TLC of 8.12 ± 0.79 in 12 dogs, and Terzungwe (2018) observed mean TLC of $7.45 \pm 2.33 \times 10^9/L$ in dogs >6 months of age with CPV infection on the day of presentation.

The significant ($P < 0.01$) increase in the TLC was seen from 0th day to 5th day in both the treatment groups, however, the increment in TLC was more prominent in T2 treated with oseltamivir. The study conducted by Savigny and Macintire (2010) on the use of oseltamivir in the treatment of canine parvoviral enteritis found that the group receiving oseltamivir showed a lack of decline in WBC count which could be a protective effect of a drug, while the group receiving placebo showed further decline WBC count. A higher WBC count could indicate a stronger immune system and a greater potential to protect against sepsis-related complications indicating the beneficial effect of oseltamivir.

In the present study, the decrease in TLC on the day of the presentation can be attributed to the destruction of hematopoietic progenitor cells of the various leukocyte types primarily in the bone marrow, but also in other lymphoproliferative organs such as the thymus, lymph nodes, and spleen, resulting in inadequate compensation for the massive demand for leukocytes (Goddard *et al.* 2008 and Alves *et al.* 2020). The TLC increased on the fifth day after treatment, indicating that antibiotic therapy enhanced immune status, whereas in T2, the significant rise in TLC could be attributed to the beneficial effect of oseltamivir.

4.6.5 Neutrophils

The mean Neutrophils ($10^3/\mu l$) in both treatment groups at scheduled intervals is tabulated in Table 4.14 and graphically depicted in Fig 4.10.

Table 4.14 Mean Neutrophils of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	67.58±4.97	73.15±2.00	70.36±2.69
	4-6	70.73±8.45	76.90±3.01	73.81±4.37
	7-9	79.03±2.77	75.11±1.28	77.07±1.57
	10-12	75.56±2.50	72.53±1.94	74.05±1.57
	Pooled mean for age, T1	73.22±2.61	74.42±1.06	73.82±1.39
T2	0-3	67.58±4.83	70.05±2.44	68.81±2.61
	4-6	74.26±8.41	72.41±2.95	73.34±4.25
	7-9	78.66±4.27	73.3±2.30	75.85±2.46
	10-12	78.98±3.68	70.21±1.01	74.60±2.25
	Pooled mean for age, T2	74.87±2.78	71.42±1.10	73.15±1.50

Critical Difference (C.D.): For Group: NS, For Age: NS, For Interval: NS

Neutrophils are the most common leukocyte in dog blood, and they are responsible for killing bacteria, fungi, yeast, algae, parasites, and viruses, as well as inducing antibody-dependent cellular cytotoxicity (Goddard *et al.* 2008).

The mean Neutrophils varied non-significantly between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 67.58±4.97, 70.73±8.45, 79.03±2.77, and 75.56±2.50, respectively, and the corresponding values on 5th day of treatment were 73.15±2.00, 76.90±3.01, 75.11±1.28, and 72.53±1.94, respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 67.58±4.83, 74.26±8.41, 78.66±4.27, and 78.98±3.68, respectively, and the corresponding values on the 5th day of treatment were 70.05±2.44, 72.41±2.95, 73.3±2.30, and 70.21±1.01, respectively.

The non-significant differences were recorded between the periodic interval (0th and 5th day) for neutrophils amongst the age group for both the treatment groups, however in T2 neutrophil count decreased on the 5th day from 0th day. The overall, observation revealed that there were non-significant differences in neutrophils in both the treatment group on 0th as well as on 5th day.

Table 4.15 Analysis of variance for Neutrophils levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	617.580	123.516	-
Factor A	1	10.935	10.935	0.107
Factor B	3	593.710	197.903	1.940
Factor C	1	30.375	30.375	0.298
A x B	3	15.653	5.218	0.051
A x C	1	129.270	129.270	1.267
B x C	3	440.041	146.680	1.438
A x B x C	3	35.272	11.757	0.115
Error	75	7650.314	102.004	-
Total	95	9523.150	-	-

The findings of the present study comply with Sagar *et al.* (2008), Roy *et al.* (2010), Dongre *et al.* (2015), Bhargavi *et al.* (2017), Mehta *et al.* (2020), and Harizan *et al.* (2021) who recorded mean neutrophils (%) as 73.2 ± 2.04 , 72 ± 0.79 , 78.0 ± 0.41 , 77.45 ± 1.29 , 73.06 ± 5.45 and 72.39 ± 0.35 respectively, in dogs with CPV infection on the day of presentation. The results were in partial agreement with Kataria *et al.* (2020) who reported an increased mean neutrophil (%) of 84.23 ± 1.27 and Ahmad *et al.* 2020 who reported a decrease in neutrophils below 65% on the day of presentation.

The current study showed a non-significant decrease in the neutrophil count after treatment as observed by Kataria *et al.* (2020). The neutropenia in CPV infection can be attributed to the destruction of mitotically active myeloblasts in bone marrow as a direct effect of the virus and may also be related to endotoxemia and possible sepsis leading to neutrophil margination, as well as a massive loss of neutrophils through the intestinal wall (Goddard *et al.* 2008). Whereas, increased percentage of neutrophils might be associated with secondary bacterial complications (Kataria *et al.* 2020, Harizan *et al.* 2021).

4.6.6 Lymphocytes

The mean Lymphocytes in both treatment groups at scheduled intervals is presented in Table 4.16 and graphically depicted in Fig 4.11.

Table 4.16 Mean Lymphocytes of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	26.88±5.24	17.73±1.33	22.00±2.97
	4-6	24.00±8.58	15.86±2.49	19.93±4.4
	7-9	16.66±2.58	15.38±0.76	16.02±1.30
	10-12	18.93±2.80	17.15±1.36	18.04±1.51
	Pooled mean for age, T1	21.62±2.64	16.38±0.77	19.00±1.41
T2	0-3	27.15±4.87	21.55±2.37	24.35±2.72
	4-6	21.33±8.25	20.40±1.74	20.86±4.02
	7-9	17.81±3.96	18.25±1.58	18.03±2.03
	10-12	15.86±2.77	18.85±1.00	17.35±1.47
	Pooled mean for age, T2	20.54±2.65	19.76±0.85	20.15±1.38

Critical Difference (C.D.): For Group: NS, For Age: NS, For Interval: NS

Table 4.17 Analysis of variance for Lymphocytes levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	533.110	106.622	-
Factor A	1	31.740	31.740	0.336
Factor B	3	568.020	189.340	2.004
Factor C	1	217.202	217.202	2.299
A x B	3	33.389	11.130	0.118
A x C	1	119.260	119.260	1.262
B x C	3	262.782	87.594	0.927
A x B x C	3	22.835	7.612	0.081
Error	75	7084.810	94.464	-
Total	95	8873.150	-	-

Lymphocytes are the second most abundant white blood cells in healthy dogs, and they play an important role in both humoral and cell-mediated immune responses.

The mean Lymphocytes varied non-significantly between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 26.88±5.24, 24.00±8.58, 16.66±2.58, and 18.93±2.80, respectively and the corresponding values on 5th day of treatment were 17.73±1.33, 15.86±2.49, 15.38±0.76, and 17.15±1.36, respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 27.15±4.87, 21.33±8.25, 17.81±3.96, and 15.86±2.77, respectively, and the corresponding values on the 5th day of treatment were 21.55±2.37, 20.40±1.74, 18.25±1.58, and 18.85±1.00, respectively.

The non-significant difference was recorded during the periodic interval (0th and 5th day) for lymphocytes amongst the age group for both the treatment groups. Overall, it revealed that there were no significant differences in lymphocytes in both treatment groups at 0th as well as 5th day, however, the overall differences amongst both the groups were comparable.

The findings of the present study are in compliance with Sagar *et al.* (2008), Mehta *et al.* (2020), and Harizan *et al.* (2021) who recorded similar findings, in dogs with CPV infection on the day of presentation. Terzungwe (2018) reported mean lymphocytes (%) as 23.61±5.94 in <6 months of age. The results were in partial agreement with Andrea *et al.* (2017) and Dash *et al.* (2017) who reported increased lymphocytes percentage. The findings are contradictory with Roy *et al.* (2010), and Dongre *et al.* (2015) who reported a decrease in lymphocytes percentage on the day of presentation.

The current study showed non-significant changes in lymphocytes after treatment in both the groups from 0th day to 5th day and was found within the normal range as a result of therapy. The lower levels of lymphocyte count might be due to the virus replication in the lymphoid organs resulting in lymphocytolysis or endogenous release of high concentrations of cortisol

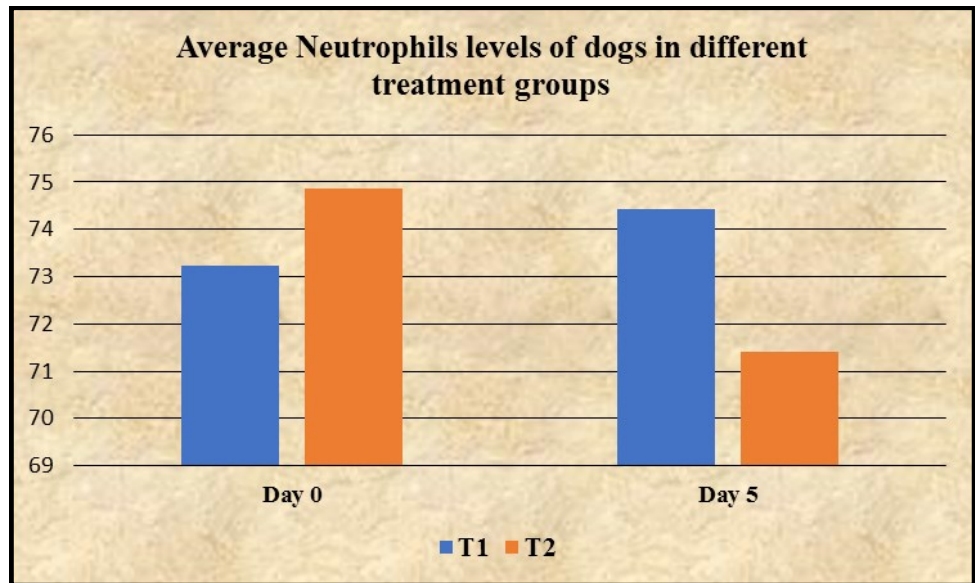


Fig. 4.10: Graphical representation of Average Neutrophils levels of dogs in different treatment groups

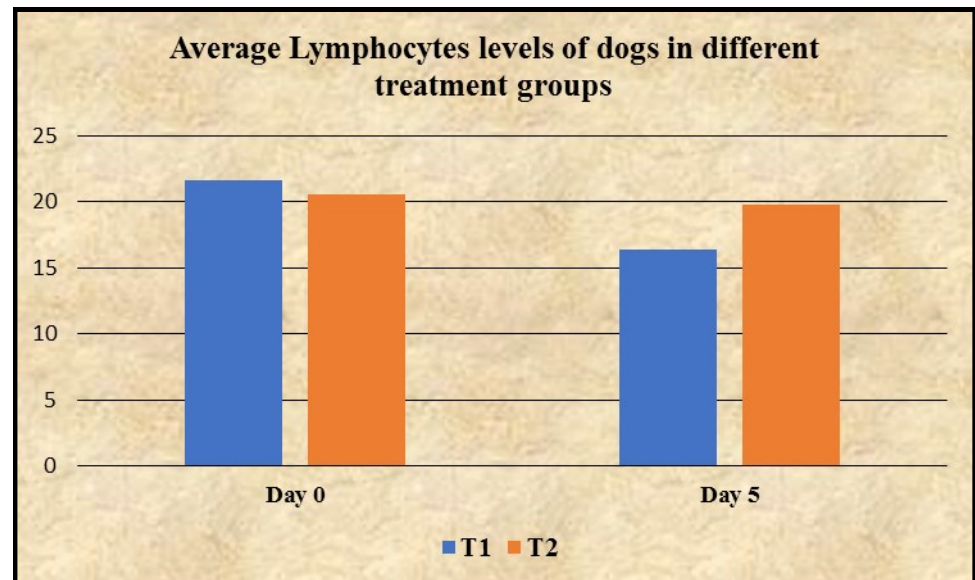


Fig. 4.11: Graphical representation of Average Lymphocytes levels of dogs in different treatment groups

(Goddard *et al.* 2008 and Kataria *et al.* 2020), while the higher levels of lymphocytes are encountered after the hyperplasia of cell lines of the bone marrow (Terzungwe 2018).

4.6.7 Eosinophils

The mean Eosinophils in both treatment groups at scheduled intervals is tabulated in Table 4.18 and graphically depicted in Fig 4.12.

The mean eosinophils varied non-significantly between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 4.65±0.91, 4.86±1.11, 4.16±0.90, and 4.40±0.96, respectively and the corresponding values on 5th day of treatment were 5.30±0.83, 4.81±0.66, 4.73±0.53, and 4.65±0.42, respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 3.73±0.55, 3.31±0.79, 5.00±0.87, and 3.46±0.66, respectively, and the corresponding values on the 5th day of treatment were 3.66±0.21, 2.53±0.24, 3.70±0.42, and 4.61±0.76, respectively.

Table 4.18 Mean Eosinophils of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	4.65±0.91	5.30±0.83	4.97±0.59
	4-6	4.86±1.11	4.81±0.66	4.84±0.62
	7-9	4.16±0.90	4.73±0.53	4.45±0.50
	10-12	4.40±0.96	4.65±0.42	4.52±0.50
	Pooled mean for age, T1	4.52±0.46	4.87±0.30	4.69±0.27
T2	0-3	3.73±0.55	3.66±0.21	3.70±0.28
	4-6	3.31±0.79	2.53±0.24	2.92±0.41
	7-9	5.00±0.87	3.70±0.42	4.35±0.50
	10-12	3.46±0.66	4.61±0.76	4.04±0.51
	Pooled mean for age, T2	3.87±0.36	3.62±0.26	3.75±0.22

Critical Difference (C.D.): For Group: 0.93, For Age: NS, For Interval: NS

Table 4.19 Analysis of variance using three-way classification of Eosinophils levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	30.816	6.163	-
Factor A	1	21.376	21.376	7.159
Factor B	3	3.922	1.307	0.438
Factor C	1	0.065	0.065	0.022
A x B	3	11.881	3.960	1.326
A x C	1	2.190	2.190	0.733
B x C	3	5.234	1.745	0.584
A x B x C	3	5.829	1.943	0.651
Error	75	223.953	2.986	-
Total	95	305.265	-	-

The non-significant differences were recorded during the periodic interval (0th and 5th day) for Eosinophils amongst the age group for both the treatment groups. The eosinophils values on 0th day were found to be non-significantly different in both groups, whereas the eosinophils value on the 5th day was found to be significantly ($P < 0.01$) higher in T1 than T2.

The findings of the present study showed eosinophils count are within the normal range in dogs with CPV infection on the day of presentation and are in compliance with Bhargavi *et al.* (2017), Dash *et al.* (2017), Terzungwe (2018), and Mehta *et al.* (2020). The current study showed non-significant changes in eosinophils after treatment in both the groups from 0th day to 5th day, whereas, marked eosinopenia found in CPV infection could be due to a combination of myelosuppression, a lack of T lymphocytes to stimulate eosinophil synthesis by the bone marrow, and also endogenous cortisol release (Goddard *et al.* 2008).

4.6.8 Monocytes

The mean monocytes in both treatment groups at scheduled intervals is tabulated in Table 4.20 and graphically depicted in Fig 4.13.

Table 4.20 Mean monocytes of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	6.20±0.61 ^A	5.65±1.34 ^A	5.92±0.70
	4-6	5.26±0.77 ^A	6.25±1.20 ^A	5.75±0.69
	7-9	4.43±0.62 ^A	7.03±0.58 ^B	5.73±0.56
	10-12	5.36±1.23 ^A	8.33±0.52 ^B	6.85±0.78
	Pooled mean for age, T1	5.31±0.41 ^A	6.81±0.50 ^B	6.06±0.34
T2	0-3	5.45±0.69 ^A	7.51±0.42 ^B	6.48±0.59
	4-6	4.40±0.85 ^A	7.23±1.40 ^B	5.81±0.89
	7-9	3.25±0.55 ^A	7.20±0.52 ^B	5.22±0.69
	10-12	4.96±1.19 ^A	8.21±0.60 ^B	6.59±0.80
	Pooled mean for age, T2	4.51±0.46 ^A	7.45±0.39 ^B	6.02±0.37

Critical Difference (C.D.): For Group: NS, For Age: NS, For Interval: 1.22

Common capital alphabets within the respective row indicate non-significant difference

Table 4.21 Analysis of variance using three-way classification of monocytes levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	5.507	1.101	-
Factor A	1	0.034	0.034	0.007
Factor B	3	20.845	6.948	1.359
Factor C	1	122.854	122.854	24.025
A x B	3	3.808	1.269	0.248
A x C	1	13.954	13.954	2.729
B x C	3	24.771	8.257	1.615
A x B x C	3	4.305	1.435	0.281
Error	75	383.523	5.114	-
Total	95	579.600	-	-

Monocytes are members of the mononuclear phagocyte system, and as macrophages, they are responsible for phagocytosis and digestion of cellular debris, bacteria, and particulate matter, as well as production of inflammatory mediators and antigen presentation to lymphocytes (Goddard *et al.* 2008).

The mean monocytes varied non-significantly between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 6.20 ± 0.61 , 5.26 ± 0.77 , 4.43 ± 0.62 , and 5.36 ± 1.23 , respectively and the corresponding values on 5th day of treatment were 5.65 ± 1.34 , 6.25 ± 1.20 , 7.03 ± 0.58 , and 8.33 ± 0.52 , respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 5.45 ± 0.69 , 4.40 ± 0.85 , 3.25 ± 0.55 , and 4.96 ± 1.19 , respectively, and the corresponding values on the 5th day of treatment were 7.51 ± 0.42 , 7.23 ± 1.40 , 7.20 ± 0.52 , and 8.21 ± 0.60 , respectively.

The significant ($P < 0.01$) differences were recorded between the periodic interval for monocytes amongst the age groups for both the treatment groups. The monocytes show significant increment on the 5th day for all the age groups in T1 as well as T2 except 0-3 and 4-6 age groups of T1. Overall, it is revealed that monocytes were appeared to be increased in both the treatment group but are within the normal range, however, the overall differences amongst both the groups were comparable.

The findings of the present study comply with Roy *et al.* (2010), Dongre *et al.* (2015), and Bhargavi *et al.* (2017), who recorded a non-significant increase in monocytes, in dogs with CPV infection on the day of presentation. The current study showed a significant increase in monocytes after treatment in both the groups from 0th day to 5th day but within the normal range. The monocytopenia is rarely seen and has little clinical importance, however monocytes and neutrophils share a common progenitor cell, the time taken to produce a monocyte in the bone marrow (3 days) is much shorter than the time taken to produce a neutrophil (6 days). Therefore, monitoring the monocyte count in the blood is beneficial for evaluating the recovery from a leukopenic state in patients suffering from CPV infection (Goddard *et al.* 2008).

(Goddard *et al.* 2008 and Kataria *et al.* 2020), while the higher levels of lymphocytes are encountered after the hyperplasia of cell lines of the bone marrow (Terzungwe 2018).

4.6.7 Eosinophils

The mean Eosinophils in both treatment groups at scheduled intervals is tabulated in Table 4.18 and graphically depicted in Fig 4.12.

The mean eosinophils varied non-significantly between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 4.65±0.91, 4.86±1.11, 4.16±0.90, and 4.40±0.96, respectively and the corresponding values on 5th day of treatment were 5.30±0.83, 4.81±0.66, 4.73±0.53, and 4.65±0.42, respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 3.73±0.55, 3.31±0.79, 5.00±0.87, and 3.46±0.66, respectively, and the corresponding values on the 5th day of treatment were 3.66±0.21, 2.53±0.24, 3.70±0.42, and 4.61±0.76, respectively.

Table 4.18 Mean Eosinophils of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	4.65±0.91	5.30±0.83	4.97±0.59
	4-6	4.86±1.11	4.81±0.66	4.84±0.62
	7-9	4.16±0.90	4.73±0.53	4.45±0.50
	10-12	4.40±0.96	4.65±0.42	4.52±0.50
	Pooled mean for age, T1	4.52±0.46	4.87±0.30	4.69±0.27
T2	0-3	3.73±0.55	3.66±0.21	3.70±0.28
	4-6	3.31±0.79	2.53±0.24	2.92±0.41
	7-9	5.00±0.87	3.70±0.42	4.35±0.50
	10-12	3.46±0.66	4.61±0.76	4.04±0.51
	Pooled mean for age, T2	3.87±0.36	3.62±0.26	3.75±0.22

Critical Difference (C.D.): For Group: 0.93, For Age: NS, For Interval: NS

Table 4.19 Analysis of variance using three-way classification of Eosinophils levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	30.816	6.163	-
Factor A	1	21.376	21.376	7.159
Factor B	3	3.922	1.307	0.438
Factor C	1	0.065	0.065	0.022
A x B	3	11.881	3.960	1.326
A x C	1	2.190	2.190	0.733
B x C	3	5.234	1.745	0.584
A x B x C	3	5.829	1.943	0.651
Error	75	223.953	2.986	-
Total	95	305.265	-	-

The non-significant differences were recorded during the periodic interval (0th and 5th day) for Eosinophils amongst the age group for both the treatment groups. The eosinophils values on 0th day were found to be non-significantly different in both groups, whereas the eosinophils value on the 5th day was found to be significantly ($P < 0.01$) higher in T1 than T2.

The findings of the present study showed eosinophils count are within the normal range in dogs with CPV infection on the day of presentation and are in compliance with Bhargavi *et al.* (2017), Dash *et al.* (2017), Terzungwe (2018), and Mehta *et al.* (2020). The current study showed non-significant changes in eosinophils after treatment in both the groups from 0th day to 5th day, whereas, marked eosinopenia found in CPV infection could be due to a combination of myelosuppression, a lack of T lymphocytes to stimulate eosinophil synthesis by the bone marrow, and also endogenous cortisol release (Goddard *et al.* 2008).

4.6.8 Monocytes

The mean monocytes in both treatment groups at scheduled intervals is tabulated in Table 4.20 and graphically depicted in Fig 4.13.

Table 4.20 Mean monocytes of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	6.20±0.61 ^A	5.65±1.34 ^A	5.92±0.70
	4-6	5.26±0.77 ^A	6.25±1.20 ^A	5.75±0.69
	7-9	4.43±0.62 ^A	7.03±0.58 ^B	5.73±0.56
	10-12	5.36±1.23 ^A	8.33±0.52 ^B	6.85±0.78
	Pooled mean for age, T1	5.31±0.41 ^A	6.81±0.50 ^B	6.06±0.34
T2	0-3	5.45±0.69 ^A	7.51±0.42 ^B	6.48±0.59
	4-6	4.40±0.85 ^A	7.23±1.40 ^B	5.81±0.89
	7-9	3.25±0.55 ^A	7.20±0.52 ^B	5.22±0.69
	10-12	4.96±1.19 ^A	8.21±0.60 ^B	6.59±0.80
	Pooled mean for age, T2	4.51±0.46 ^A	7.45±0.39 ^B	6.02±0.37

Critical Difference (C.D.): For Group: NS, For Age: NS, For Interval: 1.22

Common capital alphabets within the respective row indicate non-significant difference

Table 4.21 Analysis of variance using three-way classification of monocytes levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	5.507	1.101	-
Factor A	1	0.034	0.034	0.007
Factor B	3	20.845	6.948	1.359
Factor C	1	122.854	122.854	24.025
A x B	3	3.808	1.269	0.248
A x C	1	13.954	13.954	2.729
B x C	3	24.771	8.257	1.615
A x B x C	3	4.305	1.435	0.281
Error	75	383.523	5.114	-
Total	95	579.600	-	-

Monocytes are members of the mononuclear phagocyte system, and as macrophages, they are responsible for phagocytosis and digestion of cellular debris, bacteria, and particulate matter, as well as production of inflammatory mediators and antigen presentation to lymphocytes (Goddard *et al.* 2008).

The mean monocytes varied non-significantly between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 6.20 ± 0.61 , 5.26 ± 0.77 , 4.43 ± 0.62 , and 5.36 ± 1.23 , respectively and the corresponding values on 5th day of treatment were 5.65 ± 1.34 , 6.25 ± 1.20 , 7.03 ± 0.58 , and 8.33 ± 0.52 , respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 5.45 ± 0.69 , 4.40 ± 0.85 , 3.25 ± 0.55 , and 4.96 ± 1.19 , respectively, and the corresponding values on the 5th day of treatment were 7.51 ± 0.42 , 7.23 ± 1.40 , 7.20 ± 0.52 , and 8.21 ± 0.60 , respectively.

The significant ($P < 0.01$) differences were recorded between the periodic interval for monocytes amongst the age groups for both the treatment groups. The monocytes show significant increment on the 5th day for all the age groups in T1 as well as T2 except 0-3 and 4-6 age groups of T1. Overall, it is revealed that monocytes were appeared to be increased in both the treatment group but are within the normal range, however, the overall differences amongst both the groups were comparable.

The findings of the present study comply with Roy *et al.* (2010), Dongre *et al.* (2015), and Bhargavi *et al.* (2017), who recorded a non-significant increase in monocytes, in dogs with CPV infection on the day of presentation. The current study showed a significant increase in monocytes after treatment in both the groups from 0th day to 5th day but within the normal range. The monocytopenia is rarely seen and has little clinical importance, however monocytes and neutrophils share a common progenitor cell, the time taken to produce a monocyte in the bone marrow (3 days) is much shorter than the time taken to produce a neutrophil (6 days). Therefore, monitoring the monocyte count in the blood is beneficial for evaluating the recovery from a leukopenic state in patients suffering from CPV infection (Goddard *et al.* 2008).

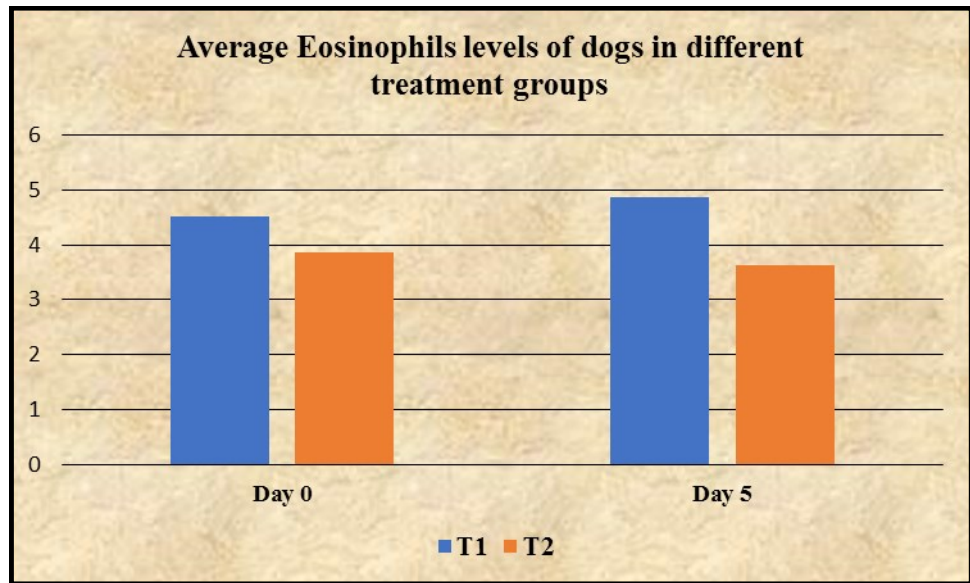


Fig. 4.12: Graphical representation of Average Eosinophils levels of dogs in different treatment groups

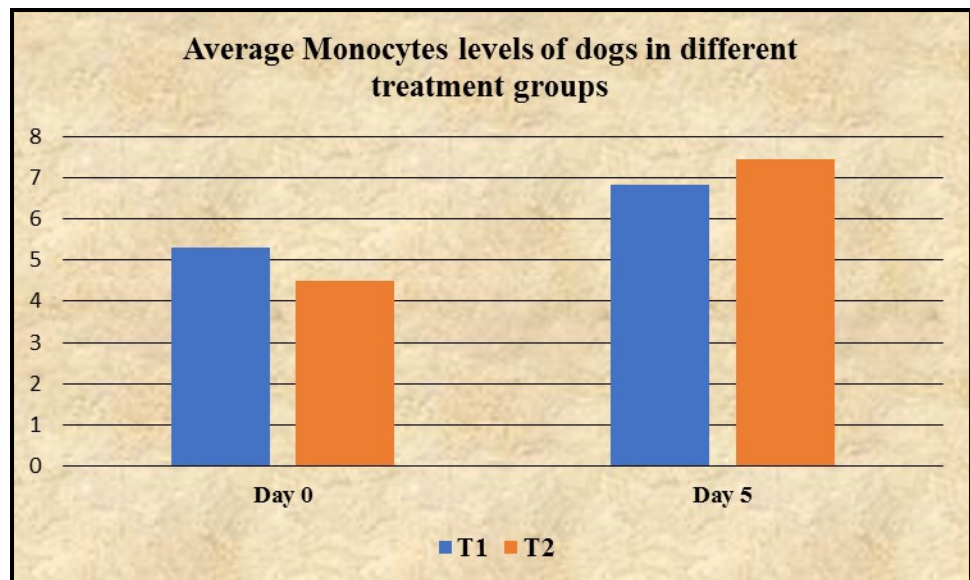


Fig. 4.13: Graphical representation of Average Monocytes levels of dogs in different treatment groups

4.6.9 CK-MB (IU/L)

The mean CK-MB (IU/L) in both treatment groups at scheduled intervals is tabulated in Table 4.22 and graphically depicted in Fig 4.14.

Table 4.22 Mean CK-MB (IU/L) of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	568.54±104.62 ^{Bc}	212.17±57.24 ^{Ab}	390.35±78.22 ^b
	4-6	512.28±76.90 ^{Bbc}	154.64±42.95 ^{Aab}	333.46±68.34 ^b
	7-9	447.65±25.38 ^{Bb}	169.96±13.50 ^{Aab}	308.81±44.04 ^b
	10-12	318.79±33.20 ^{Ba}	100.33±8.42 ^{Aa}	209.56±36.76 ^a
	Pooled mean for age, T1	461.81±37.24 ^B	159.28±19.02 ^A	310.55±30.24
T2	0-3	626.56±90.28 ^{Bc}	232.48±87.14 ^{Ab}	429.52±84.30 ^b
	4-6	430.71±72.71 ^{Bab}	164.68±35.10 ^{Aab}	297.70±55.58 ^a
	7-9	438.84±32.76 ^{Bb}	111.38±11.38 ^{Aa}	275.11±52.06 ^a
	10-12	341.27±38.91 ^{Ba}	80.47±8.49 ^{Aa}	210.87±43.66 ^a
	Pooled mean for age, T2	459.34±36.60 ^{Bc}	147.25±25.20 ^A	303.30±31.64

Critical Difference (C.D.): For Group: NS, For Age: 94.23, For Interval: 66.63

Common capital alphabets within the respective row and common small alphabets within the respective column indicate non-significant difference

CK-MB is used to assess the cardiac function in myocardial damage, such as ischemia or myocardial necrosis in both animals and humans (Gulersoy *et al.* 2020).

The mean CK-MB varied significantly ($P < 0.01$) between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 568.54±104.62, 512.28±76.90, 447.65±25.38, and 318.79±33.20, respectively and the corresponding values on 5th day of treatment were 212.17±57.24, 154.64±42.95, 169.96±13.50, and 100.33±8.42, respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were

626.56±90.28, 430.71±72.71, 438.84±32.76, and 341.27±38.91, respectively, and the corresponding values on the 5th day of treatment were 232.48±87.14, 164.68±35.10, 111.38±11.38, and 80.47±8.49, respectively.

Table 4.23 Analysis of variance using three-way classification of CK-MB (IU/L) levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	327003.647	65400.729	-
Factor A	1	1260.413	1260.413	0.083
Factor B	3	486328.908	162109.636	10.627
Factor C	1	2266592.563	2266592.563	148.580
A x B	3	22442.372	7480.791	0.490
A x C	1	547.263	547.263	0.036
B x C	3	55417.596	18472.532	1.211
A x B x C	3	20579.312	6859.771	0.450
Error	75	1144124.095	15254.988	-
Total	95	4324296.168	-	-

In T1, the pooled mean of CK-MB was observed to be 461.81 ± 37.24 on 0th day which was significantly ($P < 0.01$) higher than 159.28 ± 19.02 , observed on 5th day. In T2, the pooled mean of CK-MB was observed to be 459.34 ± 36.60 on 0th day which was significantly ($P < 0.01$) higher than 147.25 ± 25.20 , observed on 5th day. The CK-MB values on 0th day were found to be non-significantly different in both the groups, similarly, on 5th day, CK-MB values were found to be non-significantly different in both the treatment groups.

In T1, on the 0th day, the CK-MB values of 0-3 and 4-6 months age group was reported to be significantly ($P < 0.01$) greater than that of 7-9 months age group which in turn was significantly ($P < 0.01$) higher than 10-12 months age group. On 5th day, CK-MB value of 0-3 months age group was seen to be significantly ($P < 0.01$) higher than that of 4-6, 7-9, and 10-12 months age groups while the lowest mean values of CK-MB were recorded in 10-12 months age groups. In T2, on the 0th day, the mean CK-MB value of 0-3 months age group

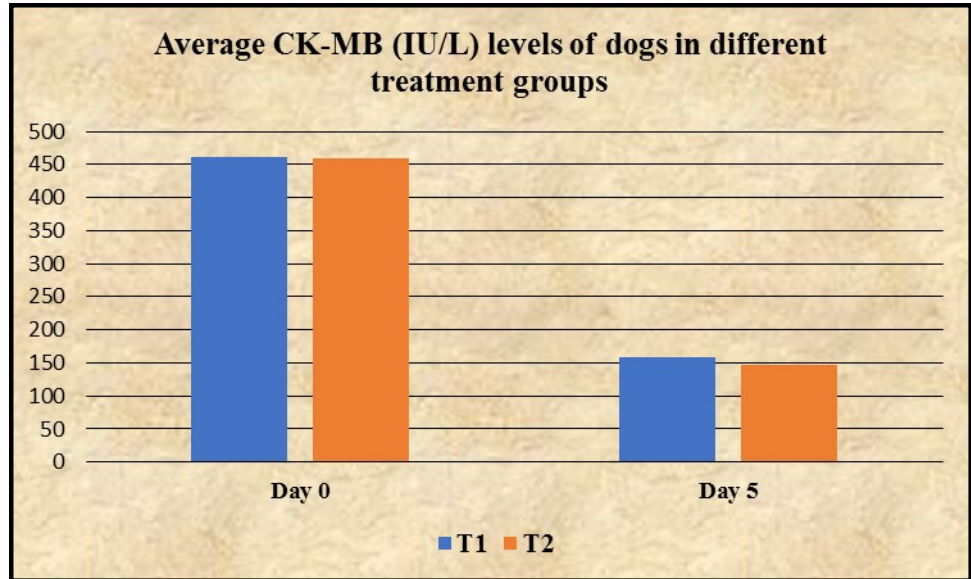


Fig. 4.14: Graphical representation of Average CK-MB levels of dogs in different treatment groups

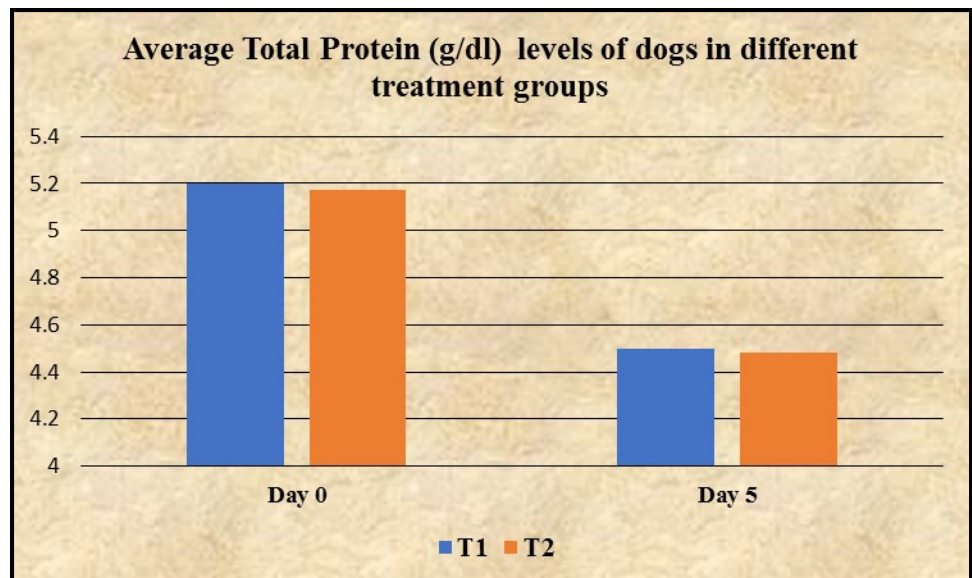


Fig. 4.15: Graphical representation of Average Total Protein levels of dogs in different treatment groups

was observed to be significantly ($P<0.01$) greater than that of 4-6 and 7-9 months age group which was observed to be statistically comparable to each other, which in the term being higher than the mean value seen in 10-12 months' age group. On 5th day, CK-MB (IU/L) value of 0-3 months age group was recorded to be significantly ($P<0.01$) higher than that seen in 4-6 and 7-9 months' age groups while the mean CK-MB value of 10-12 months age group was observed to be the lowest.

Overall, it is revealed that CK-MB was observed to be increased on the day of presentation and appeared to be decreased post-treatment in both groups. Eariler Yilmaz and Senturk (2007), Bhat *et al.* (2012), ER and OK (2015), and Gulersoy *et al.* (2020) were also reported an increased level of CK-MB in dogs with CPV infection on the day of presentation. Bhat *et al.* (2012) recorded the mean of CK-MB as 441.02 ± 36.03 in CPV infected dogs similarly observed in the current study. The present study showed a significant decrease in CK-MB (IU/L) in both the treatment groups from the 0th day to 5th day. The increased level of CK-MB indicates mild to intermediate myocarditis which may have developed in the enteritis form of CPV (ER and OK 2015 and Gulersoy *et al.* 2020). The severity of the disease was indicated by CK-MB level which may increase in blood serum rapidly, in mild to intermediate myocarditis thus it has more diagnostic value than cTnI (Bhat *et al.* 2012 and ER and OK (2015).

4.6.10 Total Protein (g/dl)

The mean Total Protein (g/dl) in both treatment groups at scheduled intervals is tabulated in Table 4.24 and graphically depicted in Fig 4.15.

The mean Total Protein varied significantly ($P<0.01$) between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 4.65 ± 0.15 , 5.35 ± 0.26 , 5.06 ± 0.09 , and 5.73 ± 0.26 , respectively and the corresponding values on 5th day of treatment were 3.98 ± 0.34 , 4.31 ± 0.22 , 4.46 ± 0.10 , and 5.23 ± 0.30 , respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 4.80 ± 0.1 , 95.18 ± 0.20 , 5.18 ± 0.31 ,

and 5.53 ± 0.28 , respectively, and the corresponding values on the 5th day of treatment were 4.21 ± 0.05 , 4.50 ± 0.12 , 4.51 ± 0.18 , and 4.71 ± 0.16 , respectively.

In T1, the pooled mean of total protein was observed to be 5.20 ± 0.12 on 0th day which was significantly ($P < 0.01$) higher than 4.50 ± 0.15 , observed on 5th day. In T2, the pooled mean of total protein was observed to be 5.17 ± 0.13 on 0th day which was significantly ($P < 0.01$) higher than 4.48 ± 0.07 , observed on 5th day. The total protein values on 0th day were found to be non-significantly different in both the groups similarly, the protein values on 5th day were found to be non-significant in both the treatment groups.

Table 4.24 Mean Total Protein (g/dl) of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	4.65 ± 0.15 ^{Ba}	3.98 ± 0.34 ^{Aa}	4.31 ± 0.20 ^a
	4-6	5.35 ± 0.26 ^{Bb}	4.31 ± 0.22 ^{Aab}	4.83 ± 0.22 ^b
	7-9	5.06 ± 0.09 ^{Bab}	4.46 ± 0.10 ^{Ab}	4.76 ± 0.11 ^b
	10-12	5.73 ± 0.26 ^{Bb}	5.23 ± 0.30 ^{Ac}	5.48 ± 0.20 ^c
	Pooled mean for age, T1	5.20 ± 0.12 ^B	4.50 ± 0.15 ^A	4.85 ± 0.11
T2	0-3	4.80 ± 0.19 ^{Ba}	4.21 ± 0.05 ^{Aa}	4.50 ± 0.12 ^a
	4-6	5.18 ± 0.20 ^{Ba}	4.50 ± 0.12 ^{Aab}	4.84 ± 0.15 ^a
	7-9	5.18 ± 0.31 ^{Ba}	4.51 ± 0.18 ^{Aab}	4.85 ± 0.20 ^a
	10-12	5.53 ± 0.28 ^{Bb}	4.71 ± 0.16 ^{Ab}	5.12 ± 0.19 ^b
	Pooled mean for age, T2	5.17 ± 0.13 ^B	4.48 ± 0.07 ^A	4.83 ± 0.09

Critical Difference (C.D.): For Group: NS, For Age: 0.41, For Interval: 0.29

Common capital alphabets within the respective row and common small alphabets within the respective column indicate non-significant difference

In T1, on the 0th day, the total protein value of 0-3 months' age group was observed to be significantly ($P < 0.01$) lower than that of 4-6, 7-9, and 10-12 months age groups. On 5th day, the total protein value of 10-12 months' age group was reported to be significantly ($P < 0.01$) higher than that seen in 4-6 and 7-9

months' age groups while the mean total protein value of 0-3 months' age group was observed to be the lowest. In T2, on the 0th day, the mean total protein value of 10-12 months' age group was observed to be significantly (P<0.01) greater than that of 0-3, 4-6, and 7-9 months' age group which were seen to be statistically comparable to each other. On 5th day, the total protein value of the 0-3 months' age group was seen to be significantly (P<0.01) lower than that observed in 4-6, 7-9, and 10-12 months' age groups. The overall observations revealed that total protein was appeared to be decreased in both the treatment group, however, the overall differences amongst both the groups were comparable.

Table 4.25 Analysis of variance for Total Protein (g/dl) levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	1.913	0.383	-
Factor A	1	0.008	0.008	0.029
Factor B	3	9.581	3.194	10.997
Factor C	1	11.551	11.551	39.773
A x B	3	1.024	0.341	1.176
A x C	1	0.001	0.001	0.003
B x C	3	0.220	0.073	0.253
A x B x C	3	0.350	0.117	0.402
Error	75	21.782	0.290	-
Total	95	46.432	-	-

The findings of the present study are in compliance with Roy *et al.* (2010), Shah *et al.*, (2013), Kumar and Kumar (2017), Kataria *et al.* (2020), El-Zahar *et al.* (2020), and Kumar *et al.* 2020 who recorded mean of total protein as 4.27±0.01, 4.68±0.58, 6.09±0.43, 5.24±0.80, 5.8±0.24, and 5.28±0.10 respectively, in dogs with CPV infection on the day of presentation. The current study showed a significant decrease in total protein in both the treatment groups from 0th day to 5th day. The decrease in the levels of total protein might be due to decreased dietary intake, decreased absorption through villi of the intestines, and protein losses during the course of the disease.

4.6.11 Albumin (g/dl)

The mean albumin (g/dl) in both treatment groups at scheduled intervals is tabulated in Table 4.26 and graphically depicted in Fig 4.16.

Table 4.26 Mean albumin (g/dl) of dogs affected with CPV infection in different treatment groups and age groups at 0th and 5th day intervals

Groups	Age	Interval (Days)		Pooled mean for groups
		0 th day	5 th day	
T1	0-3	2.27±0.19 ^{Ba}	1.81±0.09 ^{Aa}	2.04±0.12 ^a
	4-6	2.23±0.11 ^{Ba}	1.95±0.06 ^{Aa}	2.09±0.07 ^a
	7-9	2.26±0.04 ^{Ba}	1.95±0.03 ^{Aa}	2.10±0.05 ^a
	10-12	2.60±0.15 ^{Bb}	2.31±0.16 ^{Ab}	2.45±0.11 ^b
	Pooled mean for age, T1	2.34±0.07 ^B	2.00±0.06 ^A	2.17±0.52
T2	0-3	2.28±0.15 ^{Ba}	2.10±0.07 ^{Aa}	2.19±0.08 ^a
	4-6	2.30±0.11 ^{Ba}	2.06±0.06 ^{Aa}	2.18±0.07 ^a
	7-9	2.30±0.1 ^{Ba}	2.03±0.04 ^{Aa}	2.16±0.06 ^a
	10-12	2.35±0.06 ^{Ba}	2.00±0.05 ^{Aa}	2.17±0.06 ^a
	Pooled mean for age, T2	2.30±0.05 ^A	2.05±0.02 ^A	2.17±0.03

Critical Difference (C.D.): For Group: NS, For Age 0.19, For Interval: 0.29

Common capital alphabets within the respective row and common small alphabets within the respective column indicate non-significant difference

Table 4.27 Analysis of variance for albumin (g/dl) levels in dogs with CPV infection

Source of variation	Degrees of freedom	Sum of squares	Mean sum of squares	F cal
Replications	5	0.679	0.136	-
Factor A	1	0.000	0.000	0.003
Factor B	3	0.622	0.207	3.208
Factor C	1	2.121	2.121	32.812
A x B	3	0.677	0.226	3.491
A x C	1	0.036	0.036	0.563
B x C	3	0.015	0.005	0.079
A x B x C	3	0.094	0.031	0.484
Error	75	4.848	0.065	-
Total	95	9.094	-	-

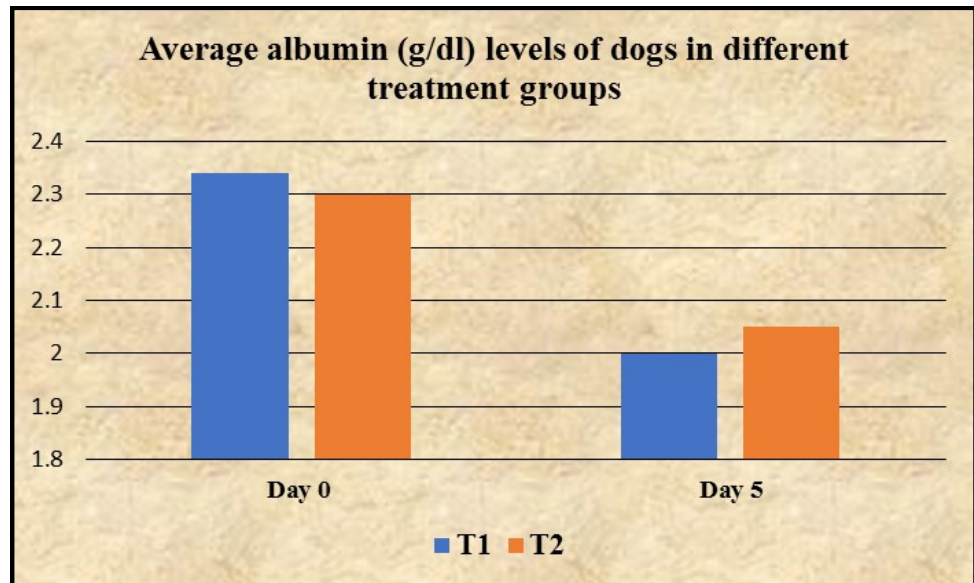


Fig. 4.16: Graphical representation of Average Albumin levels of dogs in different treatment groups

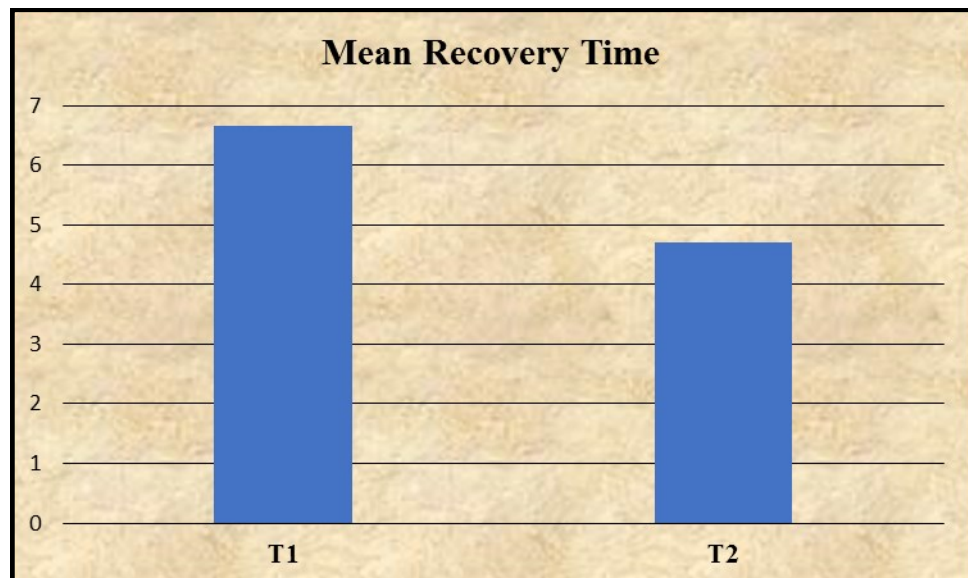


Fig. 4.17: Graphical representation of Average Recovery time of dogs in different treatment groups

The mean albumin values varied significantly ($P < 0.01$) between the different age groups, and the values of T1 for 0-3, 4-6, 7-9, and 10-12 months age group on 0th day were 2.27 ± 0.19 , 2.23 ± 0.11 , 2.26 ± 0.04 , and 2.60 ± 0.15 , respectively and the corresponding values on 5th day of treatment were 1.81 ± 0.09 , 1.95 ± 0.06 , 1.95 ± 0.03 , and 2.31 ± 0.16 , respectively. The values for 0-3, 4-6, 7-9, and 10-12 months age groups under T2 on 0th day were 2.28 ± 0.15 , 2.30 ± 0.11 , 2.30 ± 0.1 , and 2.35 ± 0.06 , respectively, and the corresponding values on the 5th day of treatment were 2.10 ± 0.07 , 2.06 ± 0.06 , 2.03 ± 0.04 , and 2.00 ± 0.05 , respectively.

In T1, the pooled mean of albumin was observed to be 2.34 ± 0.07 on 0th day which was significantly ($P < 0.01$) higher than 2.00 ± 0.06 , observed on 5th day. In T2, the pooled mean of albumin was observed to be 2.30 ± 0.05 on 0th day which was found to be non-significantly higher than 2.05 ± 0.02 , observed on 5th day. The total albumin values on 0th day were found to be non-significantly different in both the groups, similarly, on 5th day, albumin values were found to be non-significantly different in both the groups.

In T1, on the 0th day, the albumin value of 10-12 months age group was observed to be significantly ($P < 0.01$) higher than that of 0-3, 4-6, and 7-9 months age groups, however, on 5th day, albumin value of 10-12 months age group was recorded to be significantly ($P < 0.01$) higher than that seen in 0-3, 4-6 and 7-9 months age groups. In T2, on the 0th and 5th day, the mean albumin values of 0-3, 4-6, 7-9, and 10-12 months age group were observed to be statistically comparable to each other. Overall, it is revealed that albumin was appeared to be decreased in both the treatment group, however, the overall non-significant difference was observed in both the groups.

The findings of the present study are in compliance with Shah *et al.*, (2013), Salem *et al.* (2014), Acciaccia *et al.* (2019), Kumar *et al.* (2020), and Harizan *et al.* (2021). The current study showed a significant decrease in albumin in both the treatment groups from 0th day to 5th day. The decreased albumin level might be due to severe protein-losing enteropathy through the damaged capillaries

of intestinal villi and may also be due to less absorption of protein through the damaged villi.

4.7 Cardiac Troponin I Rapid Test

The 48 dogs of the present study were tested using a rapid chromatographic immunoassay-based Cardiac Troponin I Rapid Test for the qualitative detection of cardiac Troponin I, of which all the dogs were found negative (Plate 4.10). The results with Cardiac Troponin I Rapid Test kit are depicted in Plate.

The negative results of the test kit might be due to the limitation of the kit that it cannot detect less than 1.0 ng/mL of cTnI in specimens. As Bastan *et al.* (2013) recorded >0.8 ng/mL cTn-I in non-survivor and <0.156 ng/mL in survivor dogs, also cTnI level increases significantly in the first 24 to 48 h in the case of acute myocardial damage in dogs and cTnI level returns to its normal level after 48 h.

4.8 Comparative evaluation of therapeutic efficacy of conventional therapy and Oseltamivir for treatment of canine parvovirus infection.

Two groups with 24 animals each were treated with conventional therapy in T1 and therapy with Oseltamivir in T2. The efficacy of the treatment was estimated based on the ability of treatment to ameliorate clinical signs, haemato-biochemical parameters.

Estimation of haemato-biochemical parameters was done on 0th day and 5th day and reported that significant increment in leukocyte count in T2 as compared to T1 which was indicative of improved immune status and better normalization of neutrophils in T2 than T1 indicate control of secondary bacterial complications, whereas non-significant differences were observed for remaining parameters in both groups.

Clinical symptoms and signs recorded were shown to be reduced considerably in dogs belonging to both the groups T1 and T2. The dogs of the T1 group took 6.66 ± 0.19 days, whereas, dogs of the T2 group took 4.70 ± 0.16 days.

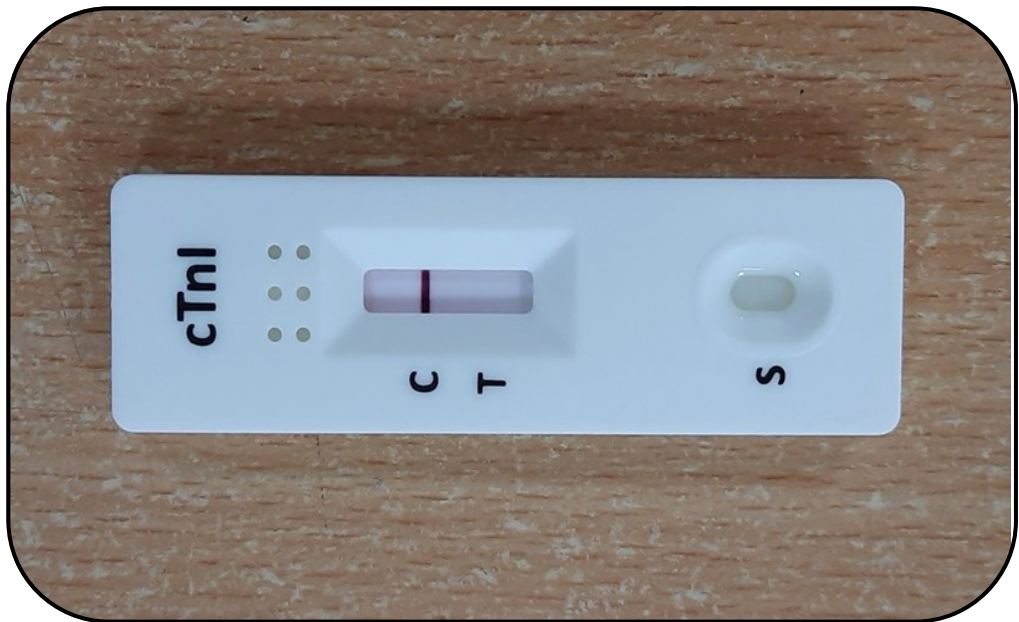


Plate 4.10: Cardiac Troponin I Rapid Test showing negative results

Based on the above-mentioned parameters and findings of the present study, it was concluded that the treatment with oseltamivir was effective in improving the leucocyte count and reducing the recovery time as compared to conventional therapy.



Plate 4.11: Infected dogs before treatment



Plate 4.12: Recovered dogs after treatment

SUMMARY AND CONCLUSION

The present study was undertaken to study the prevalence of canine parvovirus infection in dogs in Nagpur and to study the diagnosis and comparative therapeutic efficacy of antiviral drug Oseltamivir to conventional therapeutic management in canine parvovirus infection in dogs.

Diagnosis

The dogs exhibiting signs of canine parvovirus infection were subjected for identification of Parvo viral antigen in their faecal sample by using CPV Antigen Rapid Test Kit, of which 48 positive dogs were further tested by PCR and selected for the study. All faecal samples were found positive by an amplicon of 681 bp on 1% agarose gel.

Prevalence

The prevalence study of CPV infection during the period of October 2020 to September 2021 revealed that a total of 8,970 canine patients were presented at VCC, Nagpur Veterinary College, Nagpur, out of which a total of 173 dogs were diagnosed with canine parvovirus infection indicating a total of 1.93 percent affection. The age-wise prevalence was highest in young dogs, mostly less than 6 months age, on the other side adult dogs, more than a year, showed the lowest prevalence. The sex-wise prevalence of CPV infection was higher in males (58.38%) than females (41.62%). The breed-wise prevalence was found highest in non-descript dogs (35.26%), followed by Labradors (24.85%), Doberman (10.40%), German Shepherd (8.67%), Rottweiler (5.20%), Spitz (4.05%) Golden Retriever (2.89%), Lhasa Apso (2.31%), however Pug, Great Dane, Saint Bernard breeds showed an equal prevalence of 1.73%. The lowest prevalence of 1.15% was recorded in the Boxer breed. The month-wise prevalence was highest in July (23.12%), followed by August (16.76%), September (12.14%) November (11.56%), January (5.78%), April (6.94%), December (5.20%), February (4.62%), March (4.05%) and June (4.05%), however May and October showed an equal and lowest prevalence of 2.89%. The vaccination status of 48 cases revealed, 33

(68.75%) dogs were non-vaccinated for the parvovirus vaccine, 12 (25%) dogs were fully vaccinated and 3 (6.25%) dogs had taken the only first dose of vaccine.

Physical Examination

The 48 dogs diagnosed with canine parvo virus infection were examined for temperature, colour of mucous membrane and colour and consistency of faeces and vomiting. Out of 48 dogs 9 (18.75%) dogs had pyrexia, 33 (68.75%) dogs had normal rectal temperature and the rest 6 (12.5%) had subnormal body temperature. The normal mucous membrane was observed in 62.5% of dogs, pale mucous membrane in 20.83% of dogs and remaining 16.67% of dogs showed congested mucosa. The haemorrhagic diarrhoea was observed in 40 (83.33%) dogs and non-haemorrhagic diarrhoea was observed in 8 (16.67%) dogs. Considering the consistency of faeces out of 48 dogs, 45 (93.75%) dogs had watery faeces and remaining 3 (6.25%) had soft faeces on presentation. The whitish vomiting of frothy nature was observed in 11 (22.92%) dogs, plain watery type of vomiting was observed in 30 (62.5%) dogs, haematemesis was observed in 6 (12.5%) dogs and only one dog (2.08%) had yellow colour of vomiting.

Clinical Signs

The 48 dogs diagnosed with canine parvovirus infection exhibited clinical signs such as depression to lethargy, followed by inappetence (100%), pale mucosa (20.83%), fever (25%), vomition (100%), hemorrhagic diarrhoea (83.33%), non-hemorrhagic diarrhoea (16.67), and dehydration (83.33%).

Haemato-biochemical parameters

Haematological parameters were studied on the day of presentation and the 5th day. The haemoglobin, PCV, TEC, TLC, and DLC were estimated. The Hb, PCV, and TEC values were observed to be decreased on the 5th day as compared to day of presentation as a result of haemorrhagic enteritis. The increase in TLC values was observed in both the group on 5th day, however significant increase was reported in group treated with Oseltamivir. The differential leucocyte count revealed neutrophilia though non-significant changes were

observed in neutrophils, lymphocytes and eosinophils in both the groups on 0th and 5th day, however, significant changes were observed in monocytes.

Serum biochemistry values were studied on the day of presentation and the 5th day. The total protein, albumin, and creatine phosphokinase-MB were estimated. The total protein and albumin levels were found to be declined in dogs of both groups on the day of presentation indicating loss of protein during the course of the disease. After treatment on the 5th day, the total protein level further decreased significantly in both the groups whereas the albumin level in dogs belonging to group T2 showed a non-significant decrease in albumin level as compared to group T1 where it decreased significantly. The CK-MB level was elevated on the day of presentation indicating mild to intermediate myocarditis which was decreased significantly after treatment on the 5th day. Overall no significant changes were observed between-group T1 and T2 indicating the non-significant effect of oseltamivir on these parameters.

Therapeutic regimen

The dogs under the present study were divided into two groups consisting of 24 dogs each. Conventional therapy involved fluid therapy with Ringer's lactate and Dextrose normal saline as per dehydration level, antibiotic Ceftriaxone Tazobactam @ 20 mg/kg IV bid, Ondansetron @ 0.5 mg/kg bid, Pantoprazole @ 1mg/kg od, Ethamsylate @ 250-500 mg bid and amino acid preparation if required. Group T1 was treated with conventional therapy and Group T2 was given Oseltamivir @ 2 mg/kg bid along with conventional therapy.

From the investigation, following conclusion have been drawn

1. The prevalence of canine parvovirus infection at VCC during the period October 2020 to September 2021 was 1.93 percent. The highest prevalence was recorded in dogs <6 months of age. Males were found to be the most vulnerable. The Non-descript was the most commonly affected breed followed by Labrador retriever.

2. The month of July had the highest prevalence, while the months of May and October had the lowest. Non-vaccinated canines were shown to be more affected than those that had been vaccinated.
3. The CPV rapid antigen test kit can be effectively used for screening for early commencement of therapeutic regimen.
4. The PCR is one of the best diagnostic tool for confirmatory diagnosis of canine parvovirus infection.
5. The treatment with oseltamivir found to be effective in normalising leucocyte counts and lowering recovery time compared to conventional therapy.

BIBLIOGRAPHY

- Acciacca, R. A., L. A. Sullivan, T. L. Webb, V. Johnson, and S. W. Dow (2020) Clinical evaluation of hyperimmune plasma for treatment of dogs with naturally occurring parvoviral enteritis. *Journal of Veterinary Emergency and Critical Care*, 30(5); 525-533.
- Ahmad, M., and A. Waheed, (2020) Case of Canine Parvovirus Infection. *Research Journal of Veterinary Practitioners*, 8(2); 23–25.
- Aiello, S. E., M. A. Moses, and D. G. Allen (2016). *The Merck veterinary manual*.
- Albaz AZ, Sayed-Ahmed M, Younis E (2015) Investigation of the antiviral effect of acyclovir on canine parvovirus infection. *Pharmacy & Pharmacology International Journal*, 2(2);36–39. DOI: 10.15406/ppij.2015.02.00014
- Alves, F. S., F. H. Alonso, R. S. Horta, B. C. Barbosa, S. Beier, and P. R. O. Paes (2020) Prognostic values of physical and hematological parameters of dogs naturally infected with parvovirus PVC-2: retrospective study of 103 cases. *Arquivo Brasileiro de Medicina Veterinária e Zootecnia*, 72; 2127-2134.
- Andrea L., K. Vinodkumar, P.V. Tresamol, D. K. Justin, P.M. Priya (2017) Hematological Changes in Dogs with Parvovirus Enteritis in Thrissur District. *Imperial Journal of Interdisciplinary Research (IJIR)* Vol-3, Issue-6; 2017
- Appel MJ, F. W. Scott, L.E. Carmichael (1979) Isolation and immunisation studies of a canine parvo-like virus from dogs with haemorrhagic enteritis. *The Veterinary Record*. Aug;105(8);156-159. DOI: 10.1136/vr.105.8.156. PMID: 516347

- Archana, P. C., Shukla, D. K. Gupta and K. Bhoopendra (2009) Epidemiology on canine parvovirus infection. *The Indian Journal of Veterinary Research*, 18 (2); 42-44.
- Banja B. K., N. Sahoo, P. K. Das and S. K. Ray (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>
- Bastan, I., A. Kurtdede, and Ö. Z. E. N. Doğukan (2013) Prognostic usefulness of some parameters in dogs with canine parvovirus. *Ankara Üniversitesi Veteriner Fakültesi Dergisi*, 60(1); 53-58.
- Bhargavi, M., B. Shobhamani, K. Nalini Kumari and C. Srilatha (2017) Diagnostic Aspects and Haematobiochemical Changes Associated with Canine Parvoviral Enteritis in Dogs. *International Journal of Current Microbiology and Applied Sciences*, 6(11); 3357-3364.
- Bhat, A. A., D. R. Wadhwa, and M. A. Khan (2013) Therapeutic management of canine parvo viral (CPV) gastroenteritis. *Veterinary Practitioner*. 14(1); 96-97.
- Bhat, A. A., D. R. Wadhwa, S. Imran, and V. Chander (2012) Plasma concentration of creatine kinase-MB isoenzyme in dogs with intestinal form of canine parvovirus-2 infection. *Comparative Clinical Pathology*, 23(3); 665-667.
- Billett, H. H. (1990) Hemoglobin and hematocrit. *Clinical Methods: The History, Physical, and Laboratory Examinations*; 3rd edition.
- Biswas, S., D. Chakravorty, and N. R. Pradhan (2005) Clinical and haemato-biochemical changes in parvovirus infection in dogs. *Indian Journal of Veterinary Medicine*, 25(1);16-18.

- Burchell, R. K., J. P. Schoeman, A. L. Leisewitz (2014) The central role of chloride in the metabolic acid-base changes in canine parvoviral enteritis. *The Veterinary Journal*, Apr;200(1);152-6. doi: 10.1016/j.tvjl.2014.01.017. Epub 2014 Feb 4. PMID: 24613416; PMCID: PMC7128572.
- Castro, T. X., Cubel Garcia, R., Gonçalves, L. P., Costa, E. M., Marcello, G. C., Labarthe, N. V., & Mendes-de-Almeida, F. (2013). Clinical, hematological, and biochemical findings in puppies with coronavirus and parvovirus enteritis. *The Canadian veterinary journal*, 54(9), 885–888.
- Cavalli, A, G. Bozzo, N. Decaro, A. Tinelli, A. Aliberti, D. Buonavoglia (2001) Characterization of a canine parvovirus strain isolated from an adult dog. *New Microbiol.* Jul;24(3); 239-42. PMID: 11497080.
- Chethan, G. E., M. Singh, V. Chander, Akhilesh, D. Singh, J.B. Rajesh, H. Prasad and U. K. De (2021) Occurrence of Canine parvovirus-2 and Canine adenovirus-1 Infections in Dogs: A Hospital Based Study. *Indian Journal of Animal Research.* 55(2); 217-221. DOI: 10.18805/ijar. B-3936.
- D. K. Macintire, and S. Smith-Carr (1997) Canine parvovirus. Part II. Clinical signs, diagnosis and treatment. *Compendium on Continuing Education for the Practicing Veterinarian*, 19(3): pp. 291-302.
- Dash, S., M. R. Das, S. K. Senapati, G. R. Jena, and S. K. Panda (2019) *Therapeutic Alternations of Sero-biochemical Parameters and Electrolyte Concentration in Canine Parvo Virus Infection.* 8(11); 1425–1431.
- Dash, S., M. R. Das, S. Sathapathy, R. C. Patra, S. K. Senapati, and G. R. Jena (2017) Haematological, serum biochemical alterations and efficacy of different therapeutic regimens in haemorrhagic gastro-enteritis with special reference to canine parvo virus infection. *Global Journal of Biosciences and Biotechnology*, 6(2); 267-271.

- Decaro N, C. Desario, D. D. Addie, V. Martella, M. J. Vieira, G. Elia, A. Zicola, C. Davis, G. Thompson, E. Thiry, U. Truyen, C. Buonavoglia (2007) The study molecular epidemiology of canine parvovirus, Europe. *Emerging Infectious Diseases*, Aug;13(8);1222-4. doi: 10.3201/eid1308.070505. PMID: 17953097; PMCID: PMC2828098.
- Decaro, N., & Buonavoglia, C. (2012). Canine parvovirus—A review of epidemiological and diagnostic aspects, with emphasis on type 2c. *Veterinary Microbiology*, 155(1), 1–12. <https://doi.org/10.1016/j.vetmic.2011.09.007>
- Dongre J. Y., H. E. Mehta, P. A. Maheshwari (2015) Comparative evaluation of two treatment regimens against canine parvovirus infection. *Haryana Veterinarian*, 54; 83-84.
- Dongre, J., H. K. Mehta, and P. Maheswari (2013) Rapid diagnosis and clinical management of canine parvovirus infection. *Intas polivet*. 14 (1); 155-156.
- Duffy A., Dow S., Ogilvie G., Rao S., Hackett T (2010) Hematologic improvement in dogs with parvovirus infection treated with recombinant canine granulocyte- colony stimulating factor. *Journal of Veterinary Pharmacology and Therapeutics*, 33; 352–356.
- El-Zahar, H., Morsi, A., & El-Neshwy, W. (2019). Clinical, hematological and biochemical assessment of dogs naturally infected with canine parvovirus before and after therapy. *Benha Veterinary Medical Journal*, 37(1), 183–187. <https://doi.org/10.21608/bvmj.2019.18011.1110>
- ER, C., & OK, M. (2015). Levels of Cardiac Biomarkers and Coagulation Profiles in Dogs with Parvoviral Enteritis. *Kafkas Universitesi Veteriner Fakultesi Dergisi*. 21(3); 383-388. <https://doi.org/10.9775/kvfd.2014.12575>

- Esfandiari J and B. Klingeborne (2000) A comparative study of new rapid and one-step test for detection of parvo virus in faeces from dogs, cats and minks. *Journal of Veterinary Medicine-B, Infectious disease and veterinary public health*. 47(2);145-153.
- Ford, J., McEndaffer, L., Renshaw, R., Molesan, A., & Kelly, K. (2017). Parvovirus Infection Is Associated with Myocarditis and Myocardial Fibrosis in Young Dogs. *Veterinary Pathology*, 54(6), 964–971. <https://doi.org/10.1177/0300985817725387>
- Gamage, B., D. Dissanayake, D. Prasada, and I. Silva (2020) Risk, prognosis and causality of parvo viral enteritis in dogs in Sri Lanka. *Comparative Immunology, Microbiology and Infectious Diseases*, 72; 101496.
- Gaykwad, C., J. Garkhal, G. E. Chethan, S. Nandi, and U. K. De (2018) Amelioration of oxidative stress using N-acetylcysteine in canine parvoviral enteritis. *Journal of Veterinary Pharmacology and Therapeutics*, 41(1); 68-75.
- Goddard A and A. L. Leisewitz (2010) Canine Parvovirus. *Veterinary Clinics Small Animal Practice*. 40;1041-1053.
- Goddard, A., A. Leisewitz, M. Christopher, N. Duncan, and P. Becker (2008). Prognostic Usefulness of Blood Leukocyte Changes in Canine Parvoviral Enteritis. *Journal of Veterinary Internal Medicine*, 22(2); 309-316. doi:10.1111/j.1939-1676.2008.0073.x
- Gulersoy, E., M. Ok, R. Yildiz, E. Koral, M. Ider, M. Sevinc, and A. Zhunushova (2020) Assessment of intestinal and cardiac-related biomarkers in dogs with parvoviral enteritis. *Polish Journal of Veterinary Sciences*; 211-219.

- Harizan, I. M., R. K. Khinchi, S. K. Manju, and V. K. Meena (2021) Alterations in haemato-biochemical and oxidative stress indices in dogs affected with parvoviral enteritis. *The Pharma Innovation Journal*; SP-10(8): 428-431.
- Hasan, M. M., M. S. Jalal, M. Bayzid, M. A. M. Sharif, and M. Masduzzaman (2017) A Comparative Study on Canine Parvovirus Infection of Dog in Bangladesh and India. *Bangladesh Journal of Veterinary Medicine*, 14(2); 237–241. <https://doi.org/10.3329/bjvm.v14i2.31403>
- Horecka, K., S. Porter, E. Amirian, and E. Jefferson (2020) A Decade of Treatment of Canine Parvovirus in an Animal Shelter: A Retrospective Study. *Animals*, 10(6); e939.
- Kataria, D., D. Agnihotri, Y. C. Bangar, and T. Kumar (2020) Comparative evaluation of haemato-biochemical alterations in dogs regarding CPV infection. *The Pharma Innovation Journal*, 9(1); 510-513
- Khare, D. S., D. K. Gupta, P. C. Shukla, G. Das, A. Tiwari, N. S. Meena, and R. Khare (2019) Prevalence of canine parvovirus infection in dogs in Jabalpur (MP). *Journal of Entomology and Zoology Studies*; 7(3); 1495-1498
- Khare, D. S., D. K. Gupta, P. C. Shukla, G. Das, N. S. Meena, and R. Khare (2020). Clinical and haemato-biochemical changes in canine parvovirus infection. *Journal of Pharmacognosy and Phytochemistry*, 9(4); 1601-1604.
- Khatri, R. (2017) *Veterinary Science and Medical Diagnosis and Treatment of Canine Parvovirus Disease in Dogs : A Mini Review*. January. <https://doi.org/10.4172/2325-9590.1000233>
- Kumar, M., Chidri, S., & Nandi, S. (2011). A sensitive method to detect canine parvoviral DNA in faecal samples by nested polymerase chain reaction. *Indian Journal of Biotechnology*, Vol 10, April 2011, pp 183-187.

- Kumar, A., & Kumar, R. K. (2017). Clinico-hematobiochemical changes in parvo viral infection in dog. *International Journal of Science, Environment and Technology*, 6(5), 2837-2841.
- Kumar, R., Kumar, B., Kumar, S., Singh, M. K., & Kumar, R. (2020). Study of biochemical parameter in canine parvo virus infected canines. *The Pharma Innovation Journal*, 9(4), 40-43.
- Mahaprabhu, R., N. Pazhanivel, G. V. S. Rao, G. D. Raj, and M. Parthiban (2021) Prevalence of canine parvoviral enteritis in vaccinated and unvaccinated puppies in and around Chennai, Tamil Nadu, India. *The Pharma Innovation Journal*; SP-10(9); 560-563 .
- McCaw, D and Hoskins J. (2006). Canine Viral Enteritis. In: Greenne, C. Infectious Diseases of the Dogs and Cats, WB Saunders 3rd ed, Philadelphia, USA.
- Mehta, S. A., R. M. Patel, A. A. Vagh, S. V. Mavadiya, M. D. Patel, J. A. Vala, and S. M. Parmar (2017) Prevalence of Canine Parvo Viral Infection in Dogs in and around Navsari District of Gujarat State, India. *The Indian Journal of Veterinary Sciences and Biotechnology*, 13(02); 67-72.
- Mehta, S., S. Mavadiya, S. Parmar, A. Vagh, J. Vala, and R. Patel (2020) Comparative economic analysis of the treatment adopted for canine parvo virus infected dog. *Journal of Animal Research*, 10(5); 843-848.
- Merck (2010) Canine parvo virus enteritis. *The Merck Veterinary Manual*. 10th edition. Published by Merck and CO, Whitehouse Station, U.S.A; p 345-348.
- Muñoz, A.I., L. Vallejo-Castillo, A. Fragozo (2021) Increased survival in puppies affected by Canine Parvovirus type II using an immunomodulator as a therapeutic aid. *Scientific Reports*, 11; 19864. <https://doi.org/10.1038/s41598-021-99357-y>

- Nahat, F. W., M. S. Rahman, R. R. Sarker, M. K. Hassan, A. Z. Hasan, L. Akter, and M. A. Islam (2015) Prevalence of canine Parvo virus infection in street dogs using rapid antigen detection Kit. *Research in Agriculture Livestock and Fisheries*, 2(3); 459–464. <https://doi.org/10.3329/ralf.v2i3.26169>
- Nandi, S., and M. Kumar (2010) Canine parvovirus: Current perspective. In *Indian Journal of Virology* (Vol. 21, Issue 1; pp. 31–44). <https://doi.org/10.1007/s13337-010-0007-y>
- Nappert, G., E. Dunphy, D. Ruben, and F. A. Mann (2002) Determination of serum organic acids in puppies with naturally acquired parvoviral enteritis. *Canadian Journal of Veterinary Research*, 66(1); 15–18.
- Nelson, R. W., and C. G. Couto (2013) *Small Animal Internal Medicine - E-Book* (5th ed.) Elsevier Health Sciences. https://books.google.co.in/books?id=s-TXrwyo-_YC
- Nivy, R., S. Hahn, S. Perl, A. Karnieli, O. Karnieli, and I. Aroch (2011) A fatal outbreak of parvovirus infection: first detection of canine parvovirus type 2c in Israel with secondary Escherichia coli septicemia and meningoencephalitis. *Israel Journal of Veterinary Medicine*, 66(3); 96-102.
- Odueko, F. D. (2020) *Literature review on canine parvoviral enteritis variants in Nigeria*. 9(1); 26–32. <https://doi.org/10.15406/jdvar.2020.09.00274>
- Ogbu, K. I., Anene, B. M., Nweze, N. E., Okoro, J. I., DANLADI, M., & Ochai, S. O. (2017). Canine Parvovirus: A Review. *International Journal of Science And Applied Research* (ISSN: 2504-9070), 2(2), 74-95.
- Parthiban, S., H. K. Mukhopadhyaya, P.X. Antony and R. M. Pillai (2010). Epidemiology of canine parvovirus enteritis in pet dogs in Puducherry (India). *Animal Science Reporter*, 4(3).

- Parthiban, S., P. Pothiappan, and H. K. Mukhopadhyay (2015). Hematological and therapeutic aspects of canine parvovirus infection in crossbred pups. *Intas Polivet (2015) Vol. 16 (II); 445-446*
- Parthiban, S., P. Pothiappan, and H. K. Mukhopadhyay (2016) Hematological and therapeutic aspects of canine parvovirus infection in non-descript pups. *Indian Veterinary Journal, 93(1); 35-37.*
- Pereira, C. A., T. A. Monezi, D. U. Mehnert, M. D'Angelo, E. L. Durigon. (2000) Molecular characterization of canine parvovirus in Brazil by polymerase chain reaction assay. *Vet Microbiol. 75(2); 127-33. doi: 10.1016/s0378-1135(00)00214-5. PMID: 10889403.*
- Pillai, U. N., and P. M. Deepa (2010) Rapid diagnosis and treatment of canine parvoviral enteritis. *Intas Polivet. 11(II); 348-349.*
- Potgieter, L. N. D., J. B. Jone, G. S. Patton and T. A. Webb-Martin, (1981) Experimental Parvovirus infection in dogs. *Can. J. Comp. Med., 45; 212-216.*
- Prittie, J. (2004) Canine parvoviral enteritis: A review of diagnosis, management, and prevention. *Journal of Veterinary Emergency and Critical Care, 14(3); 167-176. <https://doi.org/10.1111/j.1534-6935.2004.04020.x>*
- Rabbani, A. H., Q. Ullah, O. Naseer, A. I. Raza, M. Shahid, S. Ali and Y. R. Khan (2021) Canine Parvo Virus: A Review on Current Perspectives in Seroprevalence, Diagnostics and Therapeutics. *Global Veterinaria 23 (2); 113-126.*
- Reddy, K. B., B. Shobhamani, B. Sreedevi, D. R. Prameela, and B. S. Reddy (2015). Prevalence of canine parvo viral infection in dogs in and around Tirupathi of India. *International Journal of Livestock Research, 5(3); 93-98.*

- Robinson, W. F., C. R. Huxtable, and D. A. Pass (1980). Canine parvoviral myocarditis: a morphologic description of the natural disease. *Veterinary Pathology*, 17(3); 282-293.
- Roy, S., M. Roy, and K. A. Sagar (2010). Haemato-biochemical and therapeutic studies of canine parvoviral infection. *Intas Polivet*, 11(2); 344-347.
- Sagar, A., S. Roy, and M. Roy (2008) Clinico, Haemato-Biochemical Changes and Diagnosis of Canine Parvoviral Enteritis. *Intas Polivet*, 9(2).
- Salem, N. Y. (2014) Canine viral diarrhea: clinical, hematologic and biochemical alterations with particular reference to in-clinic rapid diagnosis. *Global Veterinaria*, 13(3); 302-307.
- Savigny, M. R., and D. K. Macintire (2010) Use of oseltamivir in the treatment of canine parvoviral enteritis. *Journal of Veterinary Emergency and Critical Care*, 20(1); 132-142.
- Schoeman, J. P., Goddard, A., & Leisewitz, A. L. (2013). Biomarkers in canine parvovirus enteritis. *New Zealand Veterinary Journal*, 61(4), 217–222. <https://doi.org/10.1080/00480169.2013.776451>
- Shah, S., N. Sood, N. Wani, K. Gupta, and A. Singh (2013) Haemato-biochemical changes in canine parvoviral infection. *Indian Journal of Veterinary Pathology*, 37(2); 131–133.
- Sharma, S., P. Dhar, and M. Sharma (2019) Study of different risk factors for Canine Parvovirus infection by haemagglutination assay. *Journal of Animal Research*, 9(5); 735-739.
- Sheikh, M. O., P. M. A. Rashid, A. S. Marouf, Z. H. Raheem, S. Manjunath, and S. C. Janga (2017) Molecular typing of canine parvovirus from Sulaimani,

Iraq and Phylogenetic analysis using partial VP2 gene. *Bulgerian Journal of Veterinary Medicine*, 20(3), 225-235.

Shima, F., F. Gberindyer, M. Tion, O. Fagbohun, T. Omobowale, and H. Nottidge, (2021) Diagnostic Performance of a Rapid Immunochromatographic Test Kit for Detecting Canine Parvovirus Infection. *Topics in Companion Animal Medicine*, 45; p.100551.

Singh, D., A. K. Verma, K. Amit, S. Mukesh, S. K. Singh, A. K. Tripathi, and A. Iftekhhar (2013) Detection of Canine Parvo Virus by polymerase chain reaction assay and its prevalence in dogs in and around Mathura, Uttar Pradesh, India. *American Journal of Biochemistry and Molecular Biology*, 3(2); 264-270.

Singh, K. P., J. P. Singh, S. V. Singh, B. Singh, P. Singh, and H. N. Singh (2011) Therapeutic management of Canine Parvo virus infection. *Intas Polivet*, 12(1); 100-101.

Snedecor, G. W. and W. G. Cochran (2004) *Statistical methods*. 6th ed. New Delhi: Oxford and IBH publishing Co.

Sulthana, C. M. (2015) Clinico-pathological findings in dogs affected with canine parvo virus (CPV) infections. *Intas Polivet*, 16(2); 443-444.

Suvethika P., Kumar K. S. V. (2021) Canine parvovirus infection: A case report. *The Pharma Innovation*;10(1);141-143.

Tanwar, J., D. K. Bihani, S. Choudhary, G. Jain, and A. Chahar (2020) Prevalence of canine parvovirus infection in Bikaner (Rajasthan) by polymerase chain reaction. *Prevalence*, 100(30); 30.

- Terzungwe, T. M. (2018) Hematological parameters of dogs infected with canine parvovirus enteritis in Sumy Ukraine. *World Journal of Innovative Research*, 5(3); 262462.
- Thakur, M., & Thakur, R. (2018). Haemato-biochemical alterations in canine Parvo Virus infection affected pups. *Himachal Journal of Agricultural Research*, 43(2), 131-134.
- Tinky, S. S., R. Ambily, S. R. Nair, and M. Mini (2015) Utility of a rapid immunochromatographic strip test in detecting canine parvovirus infection compared with polymerase chain reaction. *Veterinary World*, 8(4); 523–526. <https://doi.org/10.14202/vetworld.2015.523-526>
- Vaishali, and T. Jain (2020) Canine Parvovirus Infection: A Case Report. *International Journal of Current Microbiology and Applied Sciences*, 9(2); 2993–2996. <https://doi.org/10.20546/ijcmas.2020.902.342>
- Venn, E. C., A. J. Barnes, R. J. Hansen, P. L. Boscan, D. C. Twedt, and L. A. Sullivan (2020) Serum D-Lactate Concentrations in Dogs with Parvoviral Enteritis. *Journal of Veterinary Internal Medicine*, 34(2); 691-699.
- Weiss, D. J., and Wardrop, K. J. (Eds.). (2011). *Schalm's veterinary hematology*. John Wiley and Sons.
- Yilmaz, Z., and S. Senturk (2007) Characterisation of lipid profiles in dogs with parvoviral enteritis. *Journal of Small Animal Practice*, 48(11); 643-650.
- Zhao, Y., Y. Lin, X. Zeng (2013) Genotyping and pathobiologic characterization of canine parvovirus circulating in Nanjing, China. *Virology Journal*, 10; 272. <https://doi.org/10.1186/1743-422X-10-272>

APPENDIX - I

Mean Temperature, Heart rate and Respiration Rate of dogs affected with CPV infection on day of presentation

Sr. No	Temperature	Heart Rate	Respiration Rate
1	99.3	123	33
2	101.8	158	37
3	99.6	128	39
4	102.8	186	72
5	103.1	182	52
6	101.6	182	32
7	99.4	119	37
8	102.3	124	33
9	101.6	134	34
10	102.6	126	22
11	104.9	180	24
12	104.7	144	30
13	102	112	45
14	101.9	138	39
15	104.9	140	70
16	101.4	132	42
17	100.6	128	40
18	99.8	165	52
19	101.8	134	23
20	104.5	96	36
21	100.7	141	58
22	102.4	123	30
23	103.8	163	52
24	100.3	147	48
25	99.3	126	42
26	101.6	150	44
27	101.3	144	48
28	103.3	135	20
29	102.7	142	32
30	104.1	163	59
31	102.1	162	44
32	104.6	152	45
33	102.7	132	62
34	105.4	200	72
35	103.9	128	32
36	100.9	124	34
37	100.7	112	24
38	101.2	96	32
39	102.8	152	48
40	99.8	168	61
41	102.7	190	60
42	104.4	171	61
43	101.4	123	20
44	101.6	136	31
45	100.5	148	34
46	102.7	151	41
47	100.9	143	42
48	101.6	132	34
Mean	102.08±0.23	143.438±3.39	41.70±1.96

VITA

The author **PRENAL YOGESH BADWAIK** was born in the city Tumsar in Bhandara district of Maharashtra state on Fourth of April, 1996. He completed his education from Janta High School, Tumsar and Janta Junior College, Tumsar.

He joined Nagpur Veterinary College, Nagpur in the year 2014 for Bachelor of Veterinary and Animal Husbandry degree course and graduated with First Division in 2019. The author was awarded with 'Dr. Prakash M. Belorkar Puraskaar of Gynecology, Swargiya Dr. Deb Puraskaar of Animal Genetics, Swargiya Dr. Shripad Sukalika Puraskaar of Livestock Production and Management and ' Swargiya Dr. Dinakar Dharmadhikari Smriti Puraskaar of Poultry Science' for securing highest marks in the respective subject from vidharbha region by Senior Vet Foundation in 2020 during his under graduation.

He has been actively participating in the National Cadet Corps (NCC) and has attended National Integration Camps (NIC) at Patna. After graduation, to acquire better skill-sets in disease diagnosis and treatment aspects, he enrolled for M.V.Sc degree course at Nagpur Veterinary College in 2019. During his postgraduation, he had given oral presentation at 38th ISVM Annual Convention and National Symposium on “Advancement in Veterinary Medicine in Mitigating Challenges to Animal Health” 2020, National Online Clinical Case Conference - 2020. He also attended First International E - Symposium on “Advances in Small Animal Renal Medicine” and Virtual Workshop on “Renal Critical Care for Vets” Chennai, WILDCON-2020, Nagpur, etc. The author has three research paper to his credit. He is a life member of Indian Society for Advancement of Canine Practice.

The author aims to expand his skillset for the benefit of the animals as well as the society and to continue growing as a veterinarian as well as good person.

Thesis Abstract

- a) Title of thesis : **“THERAPEUTIC MANAGEMENT OF PARVO VIRUS INFECTION IN DOGS”**
- b) Full Name of Student : **Badwaik Prenal Yogesh**
- c) Name & Address of Advisor/ Guide : **Dr. C. G. Panchbhai
Assistant Professor
Department of Veterinary Clinical Medicine
Ethics and Jurisprudence ,Nagpur
Veterinary College, Nagpur**
- d) Degree to be awarded : **MASTER OF VETERINARY SCIENCE**
- e) Year of award of degree : **2022**
- f) Major subject : **VETERINARY CLINICAL MEDICINE,
ETHICS AND JURISPRUDENCE**
- g) Total number of pages in the thesis : **80**
- h) Number of words in the thesis abstract : **300**
- i) Signature of student :
- j) Signature, Name & Address of forwarding authority :

**Associate Dean
Nagpur Veterinary College,
Nagpur**

ABSTRACT

The study entitled "THERAPEUTIC MANAGEMENT OF PARVO VIRUS INFECTION IN DOGS" was carried out in the Department of Veterinary Clinical Medicine, Ethics & Jurisprudence, Nagpur Veterinary College, Nagpur during the period of October 2020 to September 2021. Out of total 8,970 dogs reported, 173 dogs were diagnosed with canine parvovirus. The comparative therapeutic efficacy of antiviral drug Oseltamivir was evaluated. The primary

diagnosis of CPV was done using CPV rapid antigen test kit which was further confirmed by PCR. The highest prevalence of CPV was recorded in dogs <6 months of age whereas males were found most susceptible. The Non-descript and non-vaccinated dogs was predominantly affected whereas the highest prevalence was recorded in month of July.

In study of 48 dogs diagnosed with canine parvo virus infection, 9 (18.75%) dogs had pyrexia, 33 (68.75%) dogs had normal rectal temperature and the rest 6 (12.5%) had subnormal body temperature. The normal mucous membrane was observed in 62.5% of dogs, pale mucous membrane in 20.83% of dogs and remaining 16.67% of dogs showed congested mucosa. The haemorrhagic diarrhoea was observed in 40 (83.33%) dogs and non-haemorrhagic diarrhoea was observed in 8 (16.67%) dogs. The whitish vomiting of frothy nature was observed in 11 (22.92%) dogs, plain watery type of vomiting was observed in 30 (62.5%) dogs, haematemesis was observed in 6 (12.5%) dogs and only one dog (2.08%) had yellow colour of vomiting.

The 48 dogs diagnosed with canine parvovirus infection exhibited clinical signs such as depression to lethargy, followed by inappetence (100%), pale mucosa (20.83%), fever (25%), vomition (100%), haemorrhagic diarrhoea (83.33%), non-haemorrhagic diarrhoea (16.67), and dehydration (83.33%).

Forty-eight dogs were randomly selected and divided into two groups. Conventional therapy involved fluid therapy with Ringer's lactate and Dextrose normal saline, antibiotic Ceftriaxone Tazobactam, Ondansetron, Pantoprazole, Ethamsylate and amino acid preparation if required. Group T1 was treated with conventional therapy and Group T2 was given Oseltamivir along with conventional therapy. Haemato-biochemical (Hb, PCV, TEC, TLC, DLC, Total protein, albumin, CK-MB) test were performed in both the groups at 0th day and 5th day interval.

Based on haemato-biochemical parameters and improvement in clinical signs, it was observed that treatment with oseltamivir found to be effective in normalising leucocyte counts and lowering recovery time compared to conventional therapy.

प्रबंध सारांश

अ. प्रबंधाचे शर्षक	:	“श्वानामधील पार्वी वषाणू संसर्गाचे उपचारात्मक व्यवस्थापन”
ब. वद्यार्थ्यांचे पुर्ण नाव	:	बडवाईक प्रेणाल योगेश
क. मार्गदर्शकाचे नाव आ ण पत्ता	:	डॉ. सी. जी. पंचभाई सहायक प्राध्यापक पशुवैद्यक च कत्सालयीन, औषधवैद्यक शास्त्र आ ण निती व न्यायशास्त्र वभाग, नागपूर पशुवैद्यकीय महा वद्यालय, नागपूर
ड. प्रदान करण्यात येणारी पदवी	:	स्नातकोत्तर पदवी (एम. व्ही. एस. सी.)
इ. पदवी प्रदान करण्याचे वर्ष	:	२०२२
फ. मुख्य वषय	:	पशुवैद्यक च कत्सालयीन औषधवैद्यक शास्त्र आ ण निती व न्यायशास्त्र
ग. प्रबंधातील एकूण पृष्ठे	:	८०
ह. सारांशातील एकूण शब्द	:	३००
ई. वद्यार्थ्यांची सही	:	
ज. अग्रे षत करणाऱ्या अ धकाऱ्याची सही, नांव आ ण पत्ता	:	

सहयोगी अ धष्ठाता
नागपूर पशुवैद्यक महा वद्यालय,
नागपूर

सारांश

पशुवैद्यकीय च कत्सालयीन औषधशास्त्र, नीती आ ण न्यायवैद्यकशास्त्र वभाग,
नागपूर पशुवैद्यकीय महा वद्यालय, नागपूर येथे, “श्वानामधील पार्वी व्हायरस (वषाणू)

संसर्गाचे उपचारात्मक व्यवस्थापन” या वषयावर, ऑक्टोबर, २०२० ते सप्टेंबर, २०२१ या कालावधीत अभ्यास करण्यात आला. ऑक्टोबर, २०२० ते सप्टेंबर, २०२१ या कालावधीत एकूण ८९७ श्वानांचा अहवाल नोंद वला गेला. त्यांपैकी १७३ श्वानांमध्ये पार्वी वषाणू संसर्ग आढळून आला. या संसर्गग्रस्त श्वानांमध्ये ओसेलटा म वर या वषाणू वरोधी औषधाच्या तुलनात्मक परिणामकारकतेचे मूल्यांकन करण्यात आले. श्वानांमधील पार्वी व्हायरस/ वषाणू संसर्ग या आजाराचे प्राथमिक निदान हे जलद प्रतिजन चाचणी (रॅपिड अँटीजन चाचणी) द्वारे करण्यात आले, ज्याची नंतर पीसीआर द्वारे पुष्टी करण्यात आली. या आजाराचा सहा महिन्यांपेक्षा कमी वय असलेल्या श्वानांमध्ये सर्वाधिक प्रभाव नोंदवण्यात आला, ज्यामध्ये नर श्वान हे सर्वात जास्त संवेदनशील आढळून आले. या आजाराचा सर्वाधिक प्रभाव जुलै महिन्यामध्ये आढळून आला, ज्यामध्ये देशी श्वान आणि लसीकरण न झालेले श्वान मुख्यतः प्रभावित झाले होते.

पार्वी वषाणू संसर्ग ग्रस्त अभ्यासातून असलेल्या ४८ श्वानांपैकी ९ (१८.७५%) श्वानांना ज्वर होता, ३३(६८.७५%) श्वानांच्या शरीराचे तापमान सामान्य होते, आणि उर्वरित ६ (१२.५%) श्वानांच्या शरीराचे तापमान असामान्य होते. प्रभावित ४८ श्वानांपैकी ६२.५% श्वानांमध्ये श्लेष्मल त्वचा सामान्य होती, २०.८३% श्वानांमध्ये श्लेष्मल त्वचेचा रंग फक्त आणि उर्वरित १६.६७% श्वानांमध्ये रक्तसंचित श्लेष्मल त्वचा आढळून आली. ४० (८३.३३%) श्वानांमध्ये रक्तमिश्रित अतिसार/अतिसारमध्ये रक्तस्राव होता आणि उर्वरित ८ (१६.६७%) श्वानांमध्ये रक्तवरील अतिसार आढळून आला. पांढरी, फेसाळ प्रकारच्या उलट्या ११(२२.९२%) श्वानांमध्ये, ३० (६२.५%) श्वानांमध्ये पाण्यासारख्या उलट्या, ६(१२.५%) श्वानांमध्ये रक्तमिश्रित उलट्या आणि फक्त १(२.०८%) श्वानांमध्ये पवळ्या रंगाचा उलट्या आढळून आल्या.

श्वानांमधील पार्वी व्हायरस/ वषाणू संसर्ग या आजाराचे ग्रस्त, अभ्यासातून असलेल्या ४८ श्वानांमध्ये सुरुवातीला उदासीनता/औदासीन्य, अशक्तपणा आणि त्यानंतर

भूक मंदावणे (१००%), फकट श्लेष्मल त्वचा (२०.८३%), ज्वर (२५%), उलट्या (१००%), रक्त मश्रत अतिसार (८३.३३%), रक्ता वरहित अतिसार (१६.६७%) आ ण शरीरातील पाण्याची कमतरता(८३.३३%) अशा प्रकारची लक्षणे आढळून आली.

च कत्सेमध्ये अभ्यासातील ४८ श्वानांना सहजगत्यने दोन गटात वभागण्यात आले. पारंपरिक उपचारामध्ये, रिंगर लॅक्टेट (आरएल) आ ण डेक्स्ट्रोज नॉर्मल सलाईन (डीएनएस) द्वारे फ्लुईड थेरेपी (द्रव उपचार), प्रतिजैवक - सेफ्ट्रीयक्झोन ट्याझोबॅक्टम, ऑडेन्सेट्रोन, पॅटाप्रेझोल, इथाम सलेट आ ण आवश्यकता भासल्यास अ मनो आम्ले समा वष्ट करण्यात आले. दोन गटापैकी एक गट T १ मध्ये पारंपरिक उपचार पद्धतीचा अवलंब करण्यात आला तिथेच दुसरा गट T २ मध्ये पारंपरिक उपचारासोबत ओसेलटाम वर या औषधाचा उपयोग करण्यात आला. शून्य आ ण पाचव्या दिवसाच्या अंतरावर दोन्ही गटामधील श्वानांच्या रक्ताच्या चाचण्या (एचबी, पीसीवी, टीईसी, टीएलसी, डीएलसी, टोटल प्रोटीन, अल्बुमीन, सीके - एमबी) करण्यात आल्या.

रक्ताच्या चाचण्यांचे निकाल/परिणाम आ ण लक्षणांमध्ये झालेली सुधारणा यांच्या आधारे, असे निदर्शनास आले क श्वानांमधील पार्वी वषाणू संसर्ग या आजारामध्ये फक्त पारंपरिक उपचाराच्या तुलनेत, पारंपरिक उपचार अ धक ओसेलटामी वर हे पांढऱ्या पेशींची संख्या सामान्य करण्यामध्ये आ ण रोगमुक्तता होण्याचा काळ कमी करण्यामध्ये परिणामकारक आहे.