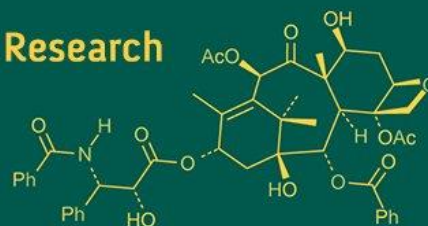
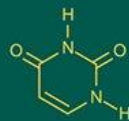


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Satish Kumar
Ph.D. Scholar, Veterinary
Medicine, Department of
Veterinary Clinical Complex,
CVASc, Pantnagar,
Uttarakhand, India

Prakash Bhatt
Professor & Head, Department
of Veterinary Clinical Complex,
CVASc, Pantnagar,
Uttarakhand, India

UK De
Senior Scientist, Indian
Veterinary Research Institute
(IVRI), Bareilly, Uttar
Pradesh, India

Mrigakshi Yadav
Veterinary Officer,
Uttarakhand State Animal
Husbandry Department,
Uttarakhand, India

Vidhi Kunwar
M.V.Sc., Veterinary Medicine,
Department of Veterinary
Clinical Complex, CVASc,
Pantnagar, Uttarakhand,
India

Corresponding Author:
Satish Kumar
Ph.D. Scholar, Veterinary
Medicine, Department of
Veterinary Clinical Complex,
CVASc, Pantnagar,
Uttarakhand, India

Evaluation of haematological alteration and oxidative stress with reference to antioxidant potential of ascorbic acid in CPV gastroenteritis

Satish Kumar, Prakash Bhatt, UK De, Mrigakshi Yadav and Vidhi Kunwar

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Abstract

Canine parvovirus type 2 (CPV-2) is a highly contagious DNA virus that primarily affects young and unvaccinated dogs. The disease is characterized by extremely high morbidity and can result in mortality rates of up to 91% when left untreated. Transmission occurs mainly through the fecal-oral route and contact with fomites contaminated with infected fecal material. Following infection, CPV-2 targets rapidly proliferating cells, particularly those in lymphoid tissues and bone marrow progenitor cells, where it undergoes extensive replication and causes significant pathological effects. Canine parvoviral disease can affect dogs of any breed or sex; however, puppies between 6 weeks and 6 months of age are particularly susceptible to infection. The clinical presentation of canine parvoviral gastroenteritis is characterized by nonspecific signs, including anorexia or decreased appetite, lethargy, weakness, depression, vomiting, dehydration, fever, and foul-smelling diarrhea that may vary from mucoid to severely hemorrhagic in nature. A total of 18 dogs were randomly selected and enrolled in the therapeutic trial. The animals were allocated into three groups (T₁, T₂, and T₃), with six dogs in each group. Group T₁ served as the healthy control group, while all dogs in Group T₂ received supportive and symptomatic treatment according to their clinical requirements. Dogs in the treatment group received a comprehensive supportive therapy regimen comprising ceftriaxone (25 mg/kg body weight, IV, twice daily for 5 days) to prevent secondary bacterial infections. Intravenous fluid therapy using Impropylte was administered to correct dehydration and electrolyte disturbances associated with vomiting and diarrhea. Metoclopramide (0.2 mg/kg body weight, IV, for 5 days) was used as an antiemetic to control vomiting, while pantoprazole (1.0 mg/kg body weight, IV, for 5 days) was administered to suppress gastric acid secretion and provide gastroprotective effects against mucosal ulceration.

Metronidazole (20 mg/kg body weight, twice daily for 5 days) was administered to manage secondary gastrointestinal infections. Botropase (0.5-1.0 mL, according to body weight) was used as a haemostatic agent until bleeding subsided. Following resolution of vomiting and diarrhea, multivitamin supplementation and a liver tonic were provided to promote hematological recovery and replenish energy reserves. Group T₃ received supportive and symptomatic therapy comparable to that of Group T₂, in addition to intravenous ascorbic acid (15 mg/kg body weight for 5 days) as an antioxidant intervention aimed at alleviating oxidative stress.

Keywords: Parvovirus, immunity, oxidative stress, gastroenteritis, canine

Introduction

Canine parvovirus type 2 (CPV-2) is a highly contagious DNA virus that primarily affects dogs, with puppies and unvaccinated animals being especially susceptible. Since its first recognition in 1967, the virus has remained a significant cause of morbidity and mortality in canine populations, exhibiting nearly 100% morbidity and mortality rates of up to 91% in the absence of appropriate treatment (Nandi and Kumar, 2010; Sykes, 2014). Canine parvovirus type 2 (CPV-2) is a small, non-enveloped virus possessing a single-stranded DNA genome. Among its variants, CPV-2a and CPV-2b are the most prevalent strains worldwide and are responsible for the majority of canine infections. Viral replication is dependent on the host cell machinery, particularly within the cell nucleus. Transmission occurs primarily through the fecal-oral route or through contact with fomites contaminated by infected fecal material. CPV-2 exhibits remarkable environmental stability, remaining infectious for up to one year under adverse conditions and surviving in contaminated soil for approximately five

months. Following entry into the host, the virus preferentially infects rapidly dividing cells, especially those located in lymphoid tissues and bone marrow progenitor cells (Nandi and Kumar, 2010) [32].

Approximately 3-4 days after infection, CPV-2 spreads hematogenously to the intestinal crypts of the small intestine. During the initial five days of viremia, the virus is widely distributed in the epithelial tissues of the oral cavity, tongue, esophagus, and small intestine, as well as in bone marrow and lymphoid organs. Infection of these rapidly proliferating cells causes cell death and impairs normal cell division, contributing to the pathogenesis of the disease (Schoeman *et al.*, 2013) [41].

Although canine parvovirus can affect dogs regardless of breed or sex, puppies between 6 weeks and 6 months of age are at the highest risk of infection (Goddard *et al.*, 2008). Long-term immunity following natural exposure or vaccination provides substantial protection in older dogs, making young puppies the most susceptible group to CPV infection (Hueffer *et al.*, 2003; Marcovich *et al.*, 2012). The clinical manifestations of canine parvoviral gastroenteritis commonly include anorexia, lethargy, weakness, depression, vomiting, dehydration, and pyrexia. Diarrhea is a prominent feature and may range from mucoid to profusely hemorrhagic, often accompanied by a characteristic foul odor (Kalli *et al.*, 2010; Tatiana *et al.*, 2013; Salem, 2014).

Canine parvovirus infection has an incubation period of approximately 4-14 days (Nandi and Kumar, 2010). While uncommon, clinical manifestations such as congestive heart failure, erythema, and neurological signs have been reported in some cases (Sykes, 2014). Hematologically, leukopenia is the hallmark abnormality associated with canine parvoviral gastroenteritis and is mainly attributable to neutropenia and/or lymphopenia caused by viral damage to bone marrow progenitor cells and lymphoid tissues (Schoeman *et al.*, 2013; Agnihotri *et al.*, 2017; Arora *et al.*, 2018). Additional hematological changes may include anemia, thrombocytopenia, thrombocytosis, pancytopenia, neutrophilic leukocytosis, and monocytosis (Kataria *et al.*, 2020; Waqas *et al.*, 2021).

In CPV-2 infections, oxidative stress and electrolyte disturbances are more closely related to disease severity than to primary causative factors (Ukwueze *et al.*, 2020). Diagnosis is often challenging due to nonspecific signs such as vomiting and diarrhea, which overlap with other gastrointestinal diseases; hence, clinical suspicion is usually confirmed using rapid antigen detection tests. Vitamin C acts as an antioxidant by scavenging free radicals (Carr and Frei, 1999), while N-acetylcysteine has been shown to improve clinical recovery and reduce hospitalization time in infected dogs (Gaykwad *et al.*, 2018). Combined use of vitamin C, N-acetylcysteine, and antibiotics has been reported to enhance recovery in CPV cases (Kataria *et al.*, 2020). Ascorbic acid, a water-soluble vitamin, acts as a major extracellular antioxidant that protects cellular components such as lipids, proteins, and DNA from oxidative injury and helps modulate inflammation. Therefore, this study was undertaken to assess the antioxidant efficacy of ascorbic acid in the treatment of CPV-associated gastroenteritis [15, 17, 29, 18, 45, 38, 32, 44, 41, 2, 4, 19].

Material and methods

The study commenced with the collection of fecal samples from dogs showing clinical signs of hemorrhagic diarrhea and vomiting. These samples were screened for canine parvovirus

using a rapid antigen detection kit (PetX, Secure Diagnostic Private Limited, Bhopal). During the study period, a total of 613 cases of hemorrhagic gastroenteritis in dogs were recorded at the Veterinary Clinical Complex, College of Veterinary and Animal Sciences, Pantnagar. Approximately 5 mL of blood was collected from each enrolled dog via cephalic vein puncture. The blood samples were divided into vials with and without EDTA. Whole blood samples were used for hematological analysis, whereas non-anticoagulated blood was allowed to clot and centrifuged at 3000 rpm for 5 minutes to obtain serum, which was subsequently stored at -20°C until further analysis.

Hematological parameters, including hemoglobin concentration, total and differential leukocyte counts, and packed cell volume (PCV), were evaluated on days 0 (pre-treatment), 3, and 5 post-treatment using standard laboratory procedures. Oxidative stress parameters, including malondialdehyde (MDA) concentration as described by Draper and Hadley (1990) serum nitric oxide (NO) levels as per Sastry *et al.* (2002) and serum ceruloplasmin concentration following the method of Sunderman and Nomoto (1970), were estimated using spectrophotometric techniques at the Clinical Diagnostic Laboratory, ICAR-IVRI, Izatnagar [12, 40, 43].

A total of 18 dogs were randomly selected for the therapeutic study and allocated into three groups (T₁, T₂, and T₃), with six animals in each group. Group T₁ served as the healthy control. Dogs in Group T₂ received supportive and symptomatic treatment as required. The treatment protocol included ceftriaxone at a dose of 25 mg/kg body weight, administered twice daily for five days to prevent secondary bacterial infections. Intravenous fluid therapy with Improlyte was provided to correct electrolyte imbalances resulting from vomiting and diarrhea. Metoclopramide was administered intravenously at 0.2 mg/kg body weight for five days to control vomiting, while pantoprazole was given intravenously at 1.0 mg/kg body weight for five days to suppress gastric acid secretion and protect the gastrointestinal mucosa from ulceration.

Metronidazole was administered at a dose of 20 mg/kg body weight twice daily for five days to manage secondary infections. A haemostatic agent was given until hemorrhage was controlled. Botropase was used as the haemostatic agent at a dose of 0.5-1.0 mL, adjusted according to the individual animal's body weight. During the recovery phase, after the resolution of vomiting and diarrhea, multivitamin supplements and a liver tonic were recommended to support hematopoietic recovery and restore energy levels as required. Group T₃ received supportive and symptomatic therapy as needed, along with intravenous ascorbic acid at a dose of 15 mg/kg body weight for five consecutive days, which was administered as antioxidant therapy to mitigate oxidative stress.

Results & Discussion

Out of 613 affected dogs included in the study, 139 were randomly selected for therapeutic evaluation. Among these, 54 dogs were confirmed positive for canine parvovirus (CPV) gastroenteritis using a rapid antigen detection test, as evidenced by the presence of both the test (T) and control (C) lines. The most common clinical manifestations observed in CPV-infected dogs were depression, anorexia, vomiting, and diarrhea.

Analysis of mean hemoglobin concentration (g/dl) revealed no significant difference between treatment groups T₂ (ST) and T₃ (ST+AA) on day 0. However, prior to the initiation of

treatment, both T₂ and T₃ exhibited significantly lower hemoglobin levels ($p < 0.05$) compared with the healthy control group (T₁).

Table 1: Effect of treatment on different haematological parameters in between group and within the group (Mean $\pm$ SE) (n=6)

Group	Days		
Hb (d/dl)	Day 0	Day 3	Day 5
T ₁	13.11 $\pm$ 0.15 ^{b_x}	13.05 $\pm$ 0.23 ^{c_x}	13.18 $\pm$ 0.30 ^{a_x}
T ₂	9.53 $\pm$ 0.50 ^{a_x}	10.06 $\pm$ 0.45 ^{a_{xy}}	11.45 $\pm$ 0.45 ^{a_y}
T ₃	8.83 $\pm$ 0.46 ^{a_x}	10.05 $\pm$ 0.48 ^{a_{xy}}	11.53 $\pm$ 0.54 ^{a_y}
PCV%			
T ₁	40.25 $\pm$ 1.00 ^{b_x}	40.23 $\pm$ 0.94 ^{b_x}	40.48 $\pm$ 1.24 ^{b_x}
T ₂	30.15 $\pm$ 1.82 ^{a_x}	33.48 $\pm$ 1.47 ^{a_{xy}}	36.61 $\pm$ 1.62 ^{ab_y}
T ₃	26.86 $\pm$ 1.64 ^{a_x}	31.06 $\pm$ 1.91 ^{a_{xy}}	34.31 $\pm$ 0.97 ^{a_y}
TLC ($\times 10^3$/cumm)			
T ₁	8.06 $\pm$ 0.17 ^{b_x}	8.01 $\pm$ 0.17 ^{b_x}	8.08 $\pm$ 0.19 ^{c_x}
T ₂	4.39 $\pm$ 0.36 ^{a_x}	4.35 $\pm$ 1.08 ^{a_x}	5.96 $\pm$ 0.93 ^{ab_x}
T ₃	3.86 $\pm$ 0.45 ^{a_x}	5.04 $\pm$ 0.34 ^{a_{xy}}	5.34 $\pm$ 0.34 ^{a_y}
Neutrophils (%)			
T ₁	76.00 $\pm$ 1.71 ^{b_x}	76.16 $\pm$ 1.24 ^{b_x}	76.16 $\pm$ 1.07 ^{b_x}
T ₂	53.66 $\pm$ 2.84 ^{a_x}	59.00 $\pm$ 2.76 ^{a_{xy}}	64.83 $\pm$ 2.41 ^{a_y}
T ₃	55.33 $\pm$ 1.85 ^{a_x}	60.16 $\pm$ 2.60 ^{a_{xy}}	67.00 $\pm$ 3.95 ^{ab_y}
Lymphocytes (%)			
T ₁	15.66 $\pm$ 1.40 ^{a_x}	15.66 $\pm$ 1.47 ^{a_x}	15.83 $\pm$ 0.87 ^{a_x}
T ₂	36.50 $\pm$ 2.87 ^{b_y}	29.83 $\pm$ 3.22 ^{b_{xy}}	24.83 $\pm$ 2.49 ^{ab_x}
T ₃	36.33 $\pm$ 1.62 ^{b_y}	32.33 $\pm$ 2.71 ^{b_{xy}}	27.00 $\pm$ 4.37 ^{b_x}
Monocytes (%)			
T ₁	7.50 $\pm$ 0.56 ^{a_x}	7.33 $\pm$ 0.49 ^{a_x}	7.16 $\pm$ 0.79 ^{a_x}
T ₂	8.83 $\pm$ 0.60 ^{a_x}	8.83 $\pm$ 0.30 ^{a_x}	8.16 $\pm$ 0.47 ^{a_x}
T ₃	7.50 $\pm$ 0.56 ^{a_x}	7.33 $\pm$ 0.49 ^{a_x}	7.33 $\pm$ 0.84 ^{a_x}
Eosinophils (%)			
T ₁	0.83 $\pm$ 0.16 ^{a_x}	0.83 $\pm$ 0.40 ^{a_x}	0.83 $\pm$ 0.47 ^{a_x}
T ₂	1.00 $\pm$ 0.25 ^{a_x}	1.00 $\pm$ 0.25 ^{a_x}	1.00 $\pm$ 0.25 ^{a_x}
T ₃	0.83 $\pm$ 0.47 ^{a_x}	0.83 $\pm$ 0.47 ^{a_x}	0.83 $\pm$ 0.47 ^{a_x}
Malonaldehyde (nM/ml)			
T ₁	5.23 $\pm$ 0.31 ^{a_x}	5.23 $\pm$ 0.31 ^{a_x}	5.23 $\pm$ 0.31 ^{a_x}
T ₂	9.81 $\pm$ 0.71 ^{b_x}	9.81 $\pm$ 0.71 ^{b_x}	9.81 $\pm$ 0.71 ^{b_x}
T ₃	8.68 $\pm$ 0.68 ^{ab_x}	8.68 $\pm$ 0.68 ^{ab_x}	8.68 $\pm$ 0.68 ^{ab_x}
Nitric oxide (μM/ml)			
T ₁	4.45 $\pm$ 0.39 ^{a_x}	4.45 $\pm$ 0.39 ^{a_x}	4.45 $\pm$ 0.39 ^{a_x}
T ₂	14.80 $\pm$ 1.19 ^{b_y}	14.80 $\pm$ 1.19 ^{b_y}	14.80 $\pm$ 1.19 ^{b_y}
T ₃	15.90 $\pm$ 0.73 ^{b_z}	15.90 $\pm$ 0.73 ^{b_z}	15.90 $\pm$ 0.73 ^{b_z}
Ceruloplasmin (g/dl)			
T ₁	0.39 $\pm$ 0.05 ^{a_x}	0.39 $\pm$ 0.05 ^{a_x}	0.39 $\pm$ 0.05 ^{a_x}
T ₂	0.78 $\pm$ 0.13 ^{b_x}	0.78 $\pm$ 0.13 ^{b_x}	0.78 $\pm$ 0.13 ^{b_x}
T ₃	0.89 $\pm$ 0.05 ^{b_y}	0.89 $\pm$ 0.05 ^{b_y}	0.89 $\pm$ 0.05 ^{b_y}

Figures with atleast one common superscript a,b,c along rows are non-significant and different subscripts x,y,z along columns differ significantly ($p < 0.05$).

By day 5, no significant differences in mean hemoglobin concentration were observed between the therapeutic groups, and their values had approached those of the healthy control group. Within-group analysis showed that the mean hemoglobin concentration (g/dl) on day 5 was significantly higher ($p < 0.05$) in both treatment groups, T₂ (ST) and T₃ (ST+AA), compared with their respective baseline values on day 0 (Table 1). All dogs in the treatment groups received supportive therapy consisting of balanced electrolyte fluids, an antiemetic (Metoclopramide @ 0.5 mg/kg body weight, IV, BID), a proton pump inhibitor (Pantoprazole @ 1 mg/kg body weight, IV, OD), antibiotics, and haemostatic agents as required. This therapeutic regimen may have helped minimize blood loss and contributed to the observed improvement in hemoglobin levels. The findings of the present study are consistent with those reported by Shah *et*

al., (2013), Terzungwe (2018), El-Zahar *et al.* (2019) and Waqas *et al.*, (2021) who also documented reduced hemoglobin concentrations and anemia in dogs affected with canine parvovirus (CPV) infection.

The observed decrease in hemoglobin concentration may be attributed to severe blood loss through hemorrhagic feces and damage to the intestinal villous capillaries caused by CPV infection. However, these findings differ from those of Gaykwad *et al.*, (2018) who reported hemoconcentration in CPV-infected dogs, which was associated with reduced water intake and excessive fluid loss resulting from vomiting and diarrhea. Furthermore, Kumar *et al.*, (2014) suggested that the gradual increase in hemoglobin concentration during recovery could be attributed to supportive therapy administered in combination with the antibiotic ceftriaxone. The mean hematocrit (%) values did not differ significantly between the therapeutic groups T₂ (ST) and T₃ (ST+AA) prior to the administration of the respective treatment protocols. However, when compared with the healthy control group

(T₁), both treatment groups exhibited significantly lower ($p < 0.05$) hematocrit values on day 0.

Following the initiation of therapy, the mean hematocrit (%) values in both T₂ (ST) and T₃ (ST+AA) showed a non-significant decrease on day 3 compared with the healthy control group (T₁). By day 5, the hematocrit value in group T₂ (ST) was comparable to that of the healthy controls, with no significant difference observed. In contrast, group T₃ (ST+AA) continued to exhibit a significantly lower ($p < 0.05$) mean hematocrit value than the healthy control group (T₁). Within-group analysis revealed that the mean hematocrit (%) values in both T₂ (ST) and T₃ (ST+AA) increased significantly ($p < 0.05$) by day 5 compared with their respective baseline values on day 0 (Table 1). The findings of the present study are consistent with those reported by El-Zahar *et al.*, (2019) who observed reduced packed cell volume (PCV) values in dogs affected with canine parvovirus (CPV), indicating the presence of anemia. However, the present results differ from those of Bhargavi *et al.*, (2017) and Kataria *et al.*, (2020) who reported elevated packed cell volume (%) in CPV-infected dogs. They attributed this increase to severe dehydration resulting from excessive fluid loss associated with diarrhea and vomiting.

The mean total leukocyte count ($\times 10^3/\text{cumm}$) was compared among the study groups. Before the initiation of treatment (day 0), the mean total leukocyte count was significantly lower ($p < 0.05$) in both therapeutic groups, T₂ (ST) and T₃ (ST+AA), compared with the healthy control group (T₁). However, no significant difference was observed between the therapeutic groups at this stage. A similar trend was noted on day 3, with no significant variation between T₂ and T₃. On day 5, although the leukocyte counts in both treatment groups remained significantly lower ($p < 0.05$) than those of the healthy control group, no significant difference was detected between the two therapeutic groups. Within-group analysis revealed that the mean leukocyte count in group T₂ (ST) did not change significantly throughout the treatment period from day 0 to day 5. In contrast, group T₃ (ST+AA) exhibited a significant increase ($p < 0.05$) in leukocyte count by day 5 following the initiation of treatment.

The findings of the present study were not in agreement with those reported by Salem (2014) and Wang and Wang (2019), who observed a significant reduction in total white blood cell (WBC), count in dogs affected with canine parvovirus (CPV) infection. Conversely, the present results are consistent with the observations of Bishnoi *et al.*, (2016), Bhargavi *et al.*, (2017), and Kataria *et al.*, (2020) who documented leukocytosis in CPV-infected dogs. The elevated leukocyte count was attributed to secondary bacterial infections resulting from damage to the intestinal epithelium caused by the viral infection [42, 46, 13, 49, 14, 26, 13, 6, 20, 38, 48, 7, 6, 20].

Comparison of the mean neutrophil (%) values among the study groups revealed that, on day 0 and day 3, both therapeutic groups T₂ (ST) and T₃ (ST+AA) had significantly lower ($p < 0.05$) neutrophil percentages than the healthy control group (T₁). However, following treatment, no significant difference in neutrophil (%) values was observed between the therapeutic groups and the healthy control group on day 5. Within-group analysis showed that the mean neutrophil (%) values in both T₂ (ST) and T₃ (ST+AA) increased significantly ($p < 0.05$) by day 5 compared with their respective baseline values on day 0 (Table 1), indicating a marked improvement following the therapeutic interventions.

The findings of the present study are consistent with those reported by Dongre *et al.*, (2015), Andrea *et al.*, (2017) and Ogbu *et al.*, (2022), who observed leukopenia accompanied by a significant decrease in neutrophil count and a corresponding increase in lymphocyte count in dogs affected with canine parvovirus infection. However, the results of the present investigation differ from those of Kalli *et al.*, (2010), Monteiro *et al.*, (2016), Agnihotri *et al.*, (2017) and Mohanta *et al.*, (2018), who documented a significant increase in neutrophil count in cases of canine parvoviral gastroenteritis. Statistical analysis of the mean lymphocyte (%) values revealed that, prior to treatment initiation (day 0) and on day 3, the diseased groups T₂ (ST) and T₃ (ST+AA) exhibited significantly higher ($p < 0.05$) lymphocyte percentages compared with the healthy control group (T₁). Following the therapeutic interventions, a significant reduction ($p < 0.05$) in lymphocyte (%) values was observed in group T₃ (ST+AA) on day 5, indicating an improvement toward normal physiological levels. Within-group comparison of lymphocyte (%) values showed a consistent pattern across the treatment period. In both T₂ (ST) and T₃ (ST+AA), the mean lymphocyte percentage was significantly reduced ($p < 0.05$) on day 5 compared with day 0 following the initiation of therapy (Table 1).

These findings are in agreement with Ramprabhu *et al.*, (2002), Kumar and Rajni (2017) and Kubesy *et al.*, (2019), who suggested that such changes may be associated with secondary bacterial infections occurring during canine parvovirus (CPV) infection. However, the present results are not consistent with those reported by Decaro and Buonavoglia (2012) and Abdullaziz *et al.*, (2022) who observed significant reductions in both total leukocyte and lymphocyte counts in CPV-infected dogs. They attributed this decline to inadequate hematopoietic activity and increased demand for blood cells due to destruction of precursor cells. In addition, Saravanan *et al.*, (2020) and Harizan *et al.*, (2021) reported that lymphopenia in CPV infection may be due to the virus's affinity for lymphoid tissues and lymphocytes, leading to their depletion.

In the present study, the mean monocyte (%) values were compared both between the therapeutic groups and within each group. It was observed that there was no significant difference in monocyte percentages among the treatment groups T₂ (ST) and T₃ (ST+AA) (Table 1). Similar findings were reported by Khare *et al.*, (2020), who also observed non-significant changes in monocyte counts. In contrast, Bastan *et al.*, (2013) reported a decrease in monocyte values in dogs affected with parvoviral infection and emphasized the beneficial role of monocytes in recovery from leukopenia in canine parvovirus gastroenteritis. This improvement was attributed to bone marrow recovery and monocyte production, which typically occurs within approximately three days.

Statistical analysis of basophil (%) values showed that, both between and within the groups, there was no significant difference observed at day 0, day 3, and day 5 (Table 1).

The findings of the present study are in agreement with those reported by Sagar *et al.*, (2008). Similarly, Khare *et al.*, (2019) also reported that there was no significant variation in basophil percentages either within or between groups from the initiation of treatment up to the stage of recovery.

The mean malondialdehyde (MDA) concentration (nM/ml) was compared among the study groups. Before the initiation of therapeutic protocols (day 0), group T₂ (ST) showed a

significantly higher ($p < 0.05$) MDA level compared with the healthy control group (T_1), whereas in group T_3 (ST+AA), the increase was non-significant. On day 3, both treatment groups T_2 and T_3 exhibited significantly elevated ($p < 0.05$) MDA levels compared with T_1 . By day 5, MDA levels remained significantly higher ($p < 0.05$) in group T_2 than in the healthy control group, while group T_3 did not differ significantly from T_1 . Furthermore, the mean MDA concentration in T_3 (ST+AA) was significantly lower ($p < 0.05$) than that observed in T_2 (ST), indicating a better reduction in oxidative stress with ascorbic acid supplementation.

The findings of the present study are in agreement with those reported by Panda *et al.*, (2009), Luo and Qiu (2013), Nykky *et al.*, (2014), Gaykwad *et al.*, (2018), and Chethan (2020), who documented increased malondialdehyde (MDA) levels (nM/ml) in dogs affected with parvovirus infection, indicating enhanced oxidative stress.

The mean nitric oxide (NO) concentration (nM/ml) was compared among the groups at different time intervals. Before the initiation of treatment (day 0) and on day 3 post-treatment, NO levels were significantly higher ($p < 0.05$) in both therapeutic groups T_2 (ST) and T_3 (ST+AA) compared with the healthy control group (T_1). On day 5, NO levels remained significantly elevated ($p < 0.05$) in group T_2 (ST) when compared with both group T_3 and the healthy control group following symptomatic treatment. In contrast, the mean NO concentration in group T_3 (ST+AA) was closer to that of the healthy control group, indicating a more pronounced normalization effect. Within-group analysis of nitric oxide (NO) levels (nM/ml) from day 0 to day 5 following therapeutic protocols revealed that there was no significant variation in NO concentration in the healthy control group (T_1) over the study period. In addition, no significant difference was observed in NO levels at day 3 compared with day 0. In contrast, group T_3 (ST+AA) showed a significant reduction ($p < 0.05$) in NO levels on day 3 post-treatment compared with day 0 (Table 1), indicating an improvement following the therapeutic intervention.

In the present study, a marked increase in nitric oxide levels was observed in dogs affected with canine parvovirus infection, which is in agreement with earlier reports by Nykky *et al.*, (2014), Gaykwad *et al.*, (2018) and Chethan (2020). These authors also reported pronounced oxidative stress in CPV-infected dogs, as evidenced by elevated NO levels prior to the initiation of treatment.

Comparison of mean NO concentrations among the treatment groups revealed a significant ($p < 0.05$) reduction in group T_3 (ST+AA) on day 3 when compared with group T_2 (ST). These findings suggest an improvement in oxidative stress status in CPV-infected dogs following antioxidant supplementation, as also reported by Gaykwad *et al.*, (2018). The mean ceruloplasmin (g/dl) levels were compared among the study groups, and it was observed that before the initiation of treatment, both therapeutic groups T_2 (ST) and T_3 (ST+AA) had significantly higher ($p < 0.05$) ceruloplasmin levels compared with the healthy control group (T_1). However, on day 5, the mean ceruloplasmin levels showed no significant difference among all groups and were comparable to the healthy control group.

Within-group analysis revealed that in group T_2 (ST), ceruloplasmin levels did not differ significantly from day 0 to day 5. In contrast, group T_3 (supportive treatment plus ascorbic acid) showed a significant reduction ($p < 0.05$) in

ceruloplasmin levels on both day 3 and day 5 following the initiation of treatment protocols (Table 1).

The findings of the present study are in agreement with Panda *et al.*, (2009) and Kubesy *et al.*, (2019), who reported that ceruloplasmin levels increase during the acute phase response in canine parvovirus (CPV) infection. They further suggested that ceruloplasmin may serve as a sensitive biomarker of inflammatory conditions. Similarly, La Rubia *et al.*, (2013) and Kocaturk *et al.*, (2015) proposed that, in addition to inflammation, the elevation of serum ceruloplasmin in CPV-infected dogs may also reflect a compensatory response against oxidative stress, owing to its antioxidant properties. In the present study, a significant ($p < 0.05$) reduction in ceruloplasmin levels was observed in groups T_3 (supportive treatment plus ascorbic acid) on day 3 post-treatment when compared with group T_2 (supportive treatment alone). This decrease may be attributed to the reduction in inflammatory status following therapeutic intervention, where passive viral neutralization contributes to decreased systemic inflammation and promotes clinical recovery.

In line with this, Kalli *et al.*, (2010) also reported a decline in serum ceruloplasmin levels following passive viral neutralization, suggesting that the reduction is associated with attenuation of the systemic inflammatory response. In CPV infection, pro-inflammatory cytokines and endotoxins play a key role in triggering and sustaining the systemic inflammatory response [11, 3, 34, 18, 31, 2, 30, 36, 25, 24, 10, 1, 39, 16, 22, 5, 37, 21, 35, 28, 33, 14, 9, 35, 24, 27, 33, 18].

Summery and conclusion

During the study period, a total of 613 cases of haemorrhagic gastroenteritis in dogs were reported at the Veterinary Clinical Complex, College of Veterinary and Animal Sciences, Pantnagar. Out of these, 139 samples were randomly selected for therapeutic evaluation, of which 54 faecal samples were confirmed positive for canine parvovirus (CPV) gastroenteritis using a rapid antigen detection test kit. Analysis of oxidative stress indices revealed a significant ($p < 0.05$) improvement, with marked reductions in malondialdehyde (MDA), nitric oxide (NO), and ceruloplasmin levels in group T_3 (supportive treatment plus ascorbic acid) compared with group T_2 (supportive treatment alone). No adverse effects associated with ascorbic acid administration were observed. The antioxidant action of ascorbic acid likely contributed to the scavenging of free radicals and mitigation of oxidative stress. Based on these findings, ascorbic acid may be considered a beneficial adjunct therapy in the management of canine parvovirus gastroenteritis.

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