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Evaluation of therapeutic efficacy of filgrastim against canine parvoviral gastroenteritis in dogs

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Abstract

The present study evaluated the use of filgrastim as an adjuvant in the treatment of canine parvovirus (CPV) infection in dogs from Mhow and surrounding areas of the Indore district, Madhya Pradesh, India. Diagnosis was conducted using the CPV Antigen Rapid Test Kit. A total of 24 CPV-positive dogs were selected and divided into four groups, each consisting of six dogs. Two groups included dogs younger than 3 months, while the other two comprised dogs aged between 3 to 6 months. Standard therapy was administered to one group in each age category, while the remaining two groups received filgrastim at a dosage of 10 µg/kg b.wt. in addition to the standard therapy. The treatment evaluation by clinical improvement, haematology and serum biochemistry were performed on day 0 of pre-treatment and day 3 and day 7 post-treatment in all the four groups. The efficacy evaluation was conducted based on the resolution of clinical signs, haematobiochemical findings, and the duration of the recovery period (in days). The therapeutic management involving filgrastim demonstrated greater efficacy compared to the conservative standard treatment, as evidenced by a more rapid recovery.

Keywords: Antigen rapid test kit, canine parvovirus (CPV), filgrastim, haematobiochemical

Introduction

Canine parvoviral gastroenteritis is one of the most common causes of morbidity and mortality in young dogs worldwide (Mylonakis *et al.*, 2016) [13]. Canine parvovirus (CPV) belongs to the genus Protoparvovirus, family Parvoviridae, a single-stranded DNA virus that infects rapidly dividing cells of the gastrointestinal tract, bone marrow, lymphoid tissue, and cardiac myocytes. CPV is found worldwide and signs of infection range from asymptomatic to severe gastroenteritis, leukopenia, dehydration, lethargy, and death (Goddard *et al.*, 2010) [6]. Puppies under 6 months of age are more commonly clinically affected, however dogs of any age can succumb to disease (Decaro *et al.*, 2012) [4]. Vaccination against CPV is routinely performed worldwide and is the most effective method to control disease (Kelman *et al.*, 2020) [8]. Total white blood cell counts and the presence of leukopenia have previously been considered to be a hallmark in patients with CPV because the virus attacks actively replicating cells of the bone marrow, thymus, and other lymphoid tissues (Castro *et al.*, 2013) [3]. The most common method for initial screening and detection of canine parvovirus is the use of a cage-side, in-clinic immunochromatographic enzyme-linked immunosorbent assay (ELISA) that has a high degree of sensitivity but intermediate to low specificity, compared with molecular methods such as PCR (Decaro *et al.*, 2012) [4]. Fluid replacement, systemic antibiotic administration, antinausea medicines, anti-diarrhoea medicines, and a rigorous diet combined with monoclonal antibodies are the main treatment methods for CPV-2-infected dogs; however, the recovery rates vary from 27.8 to 93.5% (Qi *et al.*, 2020) [16]. The destruction of haematopoietic progenitor cells of the various leukocyte types in the bone marrow and the loss of neutrophils through the injured gastrointestinal tract may cause leukopenia and neutropenia in parvo-infected dogs (Macartney *et al.*, 1984) [11]. Neutrophils are most important cells required for immune system to combat with the bacterial infection. So prolonged neutropenia greatly increases the risk of death, due to sepsis. Hence strategies are required to shorten the duration

of neutropenia in dogs. The use of granulocyte colony stimulating factor, a cytokine and growth factor that potentially stimulates neutrophil production and release from the bone marrow, has been advocated for treatment of parvovirus induced neutropenia (Kraft *et al.*, 1995) [9].

Filgrastim is a non-glycosylated, 175 amino acid containing protein, which is produced recombinantly by *Escherichia coli*. Filgrastim has a molecular weight of 18.8 kDa and it regulates the production and release of functional neutrophils from bone marrow within 24 hours of administration. Filgrastim results in increase in peripheral blood neutrophil counts with minor increase in monocytes (Areshkumar *et al.*, 2017) [1].

Materials And Methods

The study was conducted on 344 dogs diagnosed with gastroenteritis at the Veterinary Clinical Complex (VCC), College of Veterinary Science and Animal Husbandry, Mhow. The dogs were screened for canine parvovirus infection using the CPV Rapid Antigen Test Kit, an immunochromatographic assay-based diagnostic tool and 84 dogs were found to be positive for the CPV. Out of the 84 CPV positive cases, 24 dogs were selected and divided into four groups (each group comprised of six dogs and two groups had dogs less than 3 months of age and the other two groups had dogs between 3-6 months of age) and treatment was given as per the following schedule in Table 1.

Table 1: Group wise treatment protocol

Protocol		I (n=6)	II (n=6)	III (n=6)	IV (n=6)
Comparative therapeutic evaluation		< 3 months age		3-6 months age	
		Filgrastim @10 µg/kg bw S/C OD for 3 days	Placebo (Normal Saline) S/C OD	Filgrastim @10 µg/kg bw S/C OD for 3 days	Placebo (Normal Saline) S/C OD
Common treatment protocol	Antimicrobials	1. Ceftriaxone Tazobactam @ 15-25mg/kg bw IV OD 2. Metronidazole @ 10mg/kg bw IV OD			
	Anti-emetic	Ondansetron @ 0.1-0.2mg/kg bw IV OD			
	Antacid	Pantoprazole @ 1mg/kg bw IV OD			
	Haemostat	Haemocoagulase @ 0.5-1mL total dose IM or IV OD (1 CU/mL)			
	Fluid and electrolytes	Ringer's lactate and Dextrose normal saline IV as per the degree of dehydration			
	Anti-spasmodic	Dicyclomine hydrochloride @ 5-10 mg total dose IM OD			

- **Clinical evaluation:** The following parameters were assessed during the clinical examination i.e. body temperature (T), heart rate (HR), respiration rate (RR), mucous membrane and degree of dehydration.
- **Haemato-biochemical parameters:** To study haemato-biochemical parameters, 2 mL of blood was collected from either cephalic or saphenous vein using sterilized needle. 1 mL of blood was collected in vial containing Ethylenediaminetetraacetic acid (EDTA) @ 2mg/mL of blood to estimate haematological parameters and 1 mL of blood was collected in sterilized test tubes for biochemical parameters. All the above parameters were estimated on day 0th, 3rd and 7th day.
- **Statistical analysis:** The statistical analysis of data was carried out by using factorial design procedure and paired t-test interpretation based on statistical findings (Piepho *et al.*, 2018; Snedecor *et al.*, 1994) [15, 19].

Results

Among the 24 dogs included in the study, inappetence, vomiting, diarrhoea, and dehydration were observed in all cases (100%). Alterations in mucous membrane colour were also noted, with pink mucous membranes in 50% of dogs, congested mucous membranes in 25%, and pale mucous membranes in 25%. Based on the degree of dehydration, 54.17% of dogs exhibited mild dehydration, 37.50% moderate dehydration, and 8.33% severe dehydration.

The mean values of physiological parameters including heart rate and respiratory rate, were elevated on 0th day (pre-treatment) but returned to normal ranges 3rd and 7th day as shown in Table 2. Rectal temperature remained within the normal physiological range throughout the study period. However, no statistically significant differences were observed in physiological parameters during the course of treatment.

Table 2: Comparison of physical parameters before (day 0) and after (days 3 and 7) treatment

Variables		0 th day (n=6)	3 rd day (n=6)	7 th day (n=6)	Ref. Ranges
Temperature (°F)	I	101.36±0.77	101.28±0.29	101.51±0.24	99.5-102.5
	II	101.98±0.14	101.61±0.08	101.02±0.28	
	III	101.85±0.33	101.71±0.19	101.83±0.14	
	IV	101.71±0.31	101.58±0.23	101.48±0.18	
Heart rate (per min)	I	141.67 ^a ±4.41	123.33 ^b ±2.85	109.5 ^c ±3.94	70.0-120.0
	II	135.83 ^a ±2.13	118.67 ^b ±3.92	103.33 ^c ±2.71	
	III	117.33 ^a ±4.07	106.83 ^b ±4.43	102.50 ^b ±2.81	
	IV	121.67 ^a ±2.97	102.67 ^b ±2.40	96.17 ^b ±2.38	
Respiratory rate (per min)	I	40.67 ^a ±1.42	34.33 ^b ±1.60	28.83 ^c ±1.37	18.0-34.0
	II	38.67 ^a ±2.34	31.83 ^b ±3.16	27.16 ^c ±2.34	
	III	36.67 ^a ±2.49	27.51 ^b ±1.14	24.33 ^c ±0.61	
	IV	37.67 ^a ±1.58	29.67 ^b ±0.61	26.83 ^c ±0.74	

Mean bearing a, b, c superscripts differ significantly ($p \leq 0.05$) within the group in each variable.

A decreased mean total leukocyte count (TLC) was observed in all groups on 0th day, which showed a significant increase by 3rd and 7th day as clinical recovery progressed as shown in Table 3. Differential leukocyte count revealed a significant reduction in neutrophil counts in all four groups on 0th day. A significant increase in neutrophil counts was observed by 3rd

and 7th day, particularly in Group I and Group III. Lymphocyte and monocyte counts remained within normal limits throughout the study period. Platelet counts were within the normal reference range in all groups; however, a significant decrease was noted in Group I and Group III during recovery.

Table 3: Comparison of haematological parameters before (day 0) and after (days 3 and 7) treatment

Variables		0 th day (n=6)	3 rd day (n=6)	7 th day (n=6)	Ref. Ranges
Haemoglobin (g/dL)	I	13.07 ^a ±1.11	11.17 ^b ±1.12	10.02 ^b ±0.67	12.00-18.00
	II	11.96±0.87	12.08±1.03	11.36±1.01	
	III	14.31 ^a ±0.71	12.41 ^b ±0.71	10.61 ^b ±0.67	
	IV	13.79 ^a ±1.05	12.59 ^b ±0.85	12.01 ^c ±0.76	
PCV (%)	I	37.25 ^a ±2.78	31.91 ^b ±2.97	30.85 ^b ±1.77	37.00-55.00
	II	36.20±2.76	36.92±3.49	34.24±3.09	
	III	42.56 ^a ±2.38	37.20 ^b ±2.15	31.83 ^b ±2.01	
	IV	41.39 ^a ±3.15	37.78 ^b ±2.55	36.02 ^c ±2.30	
TEC (10 ⁶ /μL)	I	6.54 ^a ±0.56	5.58 ^b ±0.56	5.37 ^b ±0.35	4.95-7.87
	II	5.96±0.41	6.38±0.67	6.03±0.73	
	III	7.14 ^a ±0.35	6.19 ^b ±0.35	5.30 ^c ±0.33	
	IV	6.89 ^a ±0.52	6.29 ^b ±0.42	5.97 ^c ±0.37	
TLC (10 ³ /μL)	I	5.70 ^{aA} ±0.37	10.27 ^{bA} ±0.63	14.16 ^{cA} ±0.53	5.00-14.10
	II	6.19 ^{aA} ±0.64	8.01 ^{bB} ±0.44	8.99 ^{cB} ±0.42	
	III	5.50 ^{aC} ±0.55	8.63 ^{bC} ±0.58	13.60 ^{cC} ±0.47	
	IV	5.45 ^{aC} ±0.37	7.24 ^{bD} ±0.47	8.74 ^{cD} ±0.50	
Neutrophil (10 ³ /μL)	I	1.39 ^{aA} ±0.13	5.06 ^{bA} ±0.12	10.87 ^{cA} ±0.47	3.50-12.00
	II	2.20 ^{aA} ±0.52	3.31 ^{bB} ±0.54	4.24 ^{cB} ±0.44	
	III	1.25 ^{aC} ±0.16	4.04 ^{bC} ±0.22	10.66 ^{cC} ±0.51	
	IV	1.68 ^{aC} ±0.20	2.90 ^{bD} ±0.25	4.73 ^{cD} ±0.30	
Lymphocyte (10 ³ /μL)	I	4.21 ^a ±0.31	4.26 ^a ±0.26	3.19 ^b ±0.23	0.90-5.00
	II	3.55±0.34	4.46±0.17	4.23±0.16	
	III	3.92 ^a ±0.35	4.41 ^b ±0.31	2.87 ^c ±0.20	
	IV	3.73±0.24	4.25±0.25	3.93±0.35	
Monocyte (10 ³ /μL)	I	0.19 ^a ±0.42	0.36±0.21	0.53 ^b ±0.30	0.30-1.50
	II	0.18 ^a ±0.34	0.37±0.21	0.48 ^b ±0.30	
	III	0.17 ^a ±0.21	0.44±0.33	0.51 ^b ±0.61	
	IV	0.21 ^a ±0.34	0.39±0.25	0.46 ^b ±0.30	
Platelet (10 ³ /μL)	I	315.51 ^a ±10.86	303.16 ^b ±10.30	258.67 ^c ±11.16	200.00-500.00
	II	296.50±6.24	307.16±6.65	310.50±7.26	
	III	321.83 ^a ±12.48	307.33 ^b ±11.77	267.67 ^c ±11.67	
	IV	305.10±6.03	309.67±4.34	312.33±3.78	

PCV: Packed Cell Volume, TEC: Total Erythrocyte Count, TLC: Total Leucocyte Count. Mean bearing A, B, C, D superscripts differ significantly ($p \leq 0.05$) between the group in each variable. Mean bearing a, b, c superscripts differ significantly ($p \leq 0.05$) within the group in each variable.

All biochemical parameters remained within normal reference ranges except for total protein, albumin, and globulin, which were below normal values prior to treatment as shown in

Table 4. These parameters showed gradual improvement as treatment progressed.

Table 4: Comparison of biochemical parameters before (day 0) and after (days 3 and 7) treatment

Variables		0 th day (n=6)	3 rd day (n=6)	7 th day (n=6)	Ref. Ranges
ALT (IU/L)	I	54.37±2.76	55.93±4.73	56.56±6.30	10.0-109.0
	II	51.85±2.80	52.18±4.72	54.28±3.95	
	III	54.09±4.41	52.06±3.06	50.97±3.39	
	IV	54.41±4.99	54.93±4.24	55.45±4.94	
AST (IU/L)	I	24.31±5.10	21.29±3.90	19.57±3.01	13.0-15.0
	II	20.35±3.49	20.14±3.09	19.39±2.44	
	III	16.73±1.88	16.23±1.24	14.80±0.96	
	IV	15.70±1.20	16.04±1.41	15.34±0.88	
BUN (mg/dL)	I	18.47±2.73	18.28±2.24	17.02±1.80	12.0-25.0
	II	16.04±2.38	15.67±2.41	14.59±1.52	
	III	17.96±2.13	17.78±2.06	16.67±1.92	
	IV	17.27±1.90	17.84±1.57	18.04±1.90	
CRT (mg/dL)	I	0.84±0.10	0.82±0.06	0.83±0.07	0.5-1.5
	II	0.76±0.04	0.81±0.03	0.86±0.06	
	III	0.74±0.05	0.78±0.05	0.81±0.03	
	IV	0.89±0.08	0.92±0.08	0.95±0.08	
Total protein (g/dL)	I	5.10±0.29	5.45±0.24	5.58±0.36	5.2-8.2
	II	5.23±0.18	5.04±0.25	5.50±0.23	
	III	4.88 ^a ±0.17	5.54 ^b ±0.17	5.89 ^b ±0.15	
	IV	5.07 ^a ±0.24	5.22 ^b ±0.24	5.58 ^b ±0.26	
Albumin (g/dL)	I	2.06±0.08	2.32±0.13	2.56±0.24	2.3-4.0
	II	2.26±0.12	2.35±0.17	2.53±0.23	
	III	2.25 ^a ±0.13	2.67 ^b ±0.12	3.33 ^c ±0.18	
	IV	2.35 ^a ±0.19	2.63 ^b ±0.20	2.82 ^b ±0.18	

Globulin (g/dL)	I	3.03±0.31	3.13±0.29	3.01±0.30	2.5-4.5
	II	2.96±0.21	2.69±0.16	2.95±0.22	
	III	2.68±0.09	2.86±0.12	2.55±0.17	
	IV	2.72±0.15	2.59±0.18	2.74±0.25	

ALT: Alanine Aminotransferase, AST: Aspartate Aminotransferase, BUN: Blood Urea Nitrogen, CRT: Creatinine. Mean bearing A, B, C, D superscripts differ significantly ($p \leq 0.05$) between the group in each variable. Mean bearing a, b, c superscripts differ significantly ($p \leq 0.05$) within the group in each variable.

Dogs treated with filgrastim in combination with standard therapy exhibited a significantly shorter recovery period compared to those receiving standard treatment alone as shown in Table 5. Furthermore, total leukocyte and neutrophil counts showed significantly greater increases in Group I and Group III than in Group II and Group IV on both 3rd and 7th day post-treatment. A marked increase in total leukocyte and neutrophil counts was evident in dogs receiving granulocyte colony-stimulating factor therapy as shown in Table 3.

Table 5: Comparison of treatment groups by normalcy attained in recovery period (days)

Group	Mean total No. recovery period (days)
I	5.67
II	7.50
III	5.83
IV	7.16

Discussions

A polypeptide called cachectin, produced by activated macrophages along with interleukins, has been recognized as a major contributor to the fever and anorexia seen in inflammatory diseases. Triggered by endotoxins, cachectin is believed to be a key mediator in the development of endotoxic shock (Strombeck *et al.*, 1991) [22]. Further, decreased contractions and hypomotility has also been attributed as causes of fever and inappetence (Carman *et al.*, 1982) [2]. The inflammatory damage inflicted on the stomach and intestinal mucosal barrier is considered the underlying cause of vomiting in cases of parvoviral gastroenteritis (Mccaw *et al.*, 2006; Streck *et al.*, 2009) [12, 20]. Parvovirus infection of the germinal epithelium in the intestinal crypts leads to the breakdown and collapse of the epithelium, causing the villi to shorten. This disrupts the normal cell turnover in the small intestine. Additionally, conditions like disseminated intravascular coagulation (DIC), endotoxemia, and damage to the intestinal lining encourage secondary bacterial infections from Gram-negative and anaerobic bacteria. These intestinal changes caused by parvoviral infection are believed to contribute to the haemorrhagic diarrhoea seen in dogs with the disease. The hallmark features of parvoviral gastroenteritis in dogs are fluid and electrolyte loss resulting from persistent vomiting and diarrhoea. These symptoms are attributed to damage in the gastrointestinal epithelium, which leads to significant fluid and protein loss. Congested mucous membranes are indicative of endotoxemia in canine parvoviral gastroenteritis, potentially resulting from the invasion of Gram-negative organisms following epithelial damage. This congestion is attributed to endotoxemia and the resultant pooling of blood in capillaries and blood vessels (Strombeck *et al.*, 1991) [22].

Severe vomiting and haemorrhagic diarrhoea associated with parvoviral gastroenteritis can result in blood loss, leading to reductions in RBC count, haemoglobin concentration, and packed cell volume (Otto *et al.*, 1999) [14]. Parvovirus also targets rapidly proliferating cells in the bone marrow, inhibiting erythropoiesis and exacerbating anaemia. Initially, dehydration may cause haemoconcentration, but fluid

replacement therapy often leads to a subsequent decline in PCV due to dilutional effects (Greene *et al.*, 2012) [7].

The destruction of haematopoietic progenitor cells responsible for the production of various leukocyte types in the bone marrow, coupled with the loss of neutrophils through the damaged gastrointestinal tract, likely contributes to leukopenia and neutropenia in dogs infected with parvovirus (Macartney *et al.*, 1984) [11]. Additionally, neutropenia may arise from an overwhelming demand for neutrophils in the gut due to ongoing inflammation and infection (Ettinger *et al.*, 2016) [5].

The thrombocytopenia observed in dogs with parvoviral gastroenteritis is thought to result primarily from the destruction of megakaryocyte precursors in the bone marrow, which limits platelet production. Additionally, an increased utilization of platelets within the damaged gastrointestinal system exacerbates the depletion of circulating platelets. This dual mechanism of bone marrow suppression and increased consumption contributes to the marked reduction in platelet levels seen in affected dogs (Rewerts *et al.*, 2000) [17].

Filgrastim stimulates the production and release of neutrophils from the bone marrow, but its effects on other blood cell lineages, such as megakaryocytes (precursors to platelets), can lead to a relative decrease in platelet production. Additionally, rapid neutrophil proliferation may cause bone marrow exhaustion, limiting the availability of resources for megakaryocyte development, thereby contributing to thrombocytopenia in some cases (Wu *et al.*, 2020) [22].

Anorexia, along with reduced food intake, induces a decrease in hepatic protein synthesis as amino acids are preferentially utilized for the production of positive acute phase proteins in response to inflammatory stimuli and tissue injury. This shift is a key component of the acute phase response, where the liver alters its metabolic function to support immune activation and repair mechanisms. As a consequence, there is a decrease in the synthesis of albumin and other essential proteins, contributing to hypoproteinaemia (Li *et al.*, 2015) [10].

Numerous studies have shown the effectiveness of recombinant human granulocyte-colony stimulating factor (rhG-CSF) in treating leukopenia, particularly neutropenia, in dogs. This evidence provides strong support for the inclusion of filgrastim as an adjuvant therapy in the current study for treating canine parvoviral gastroenteritis in dogs with leukopenia (Kraft *et al.*, 1995) [9]. The role of granulocyte colony-stimulating factor (G-CSF) in modulating neutrophil function is well-established in the literature. As a key cytokine, G-CSF regulates both the generation and differentiation of neutrophils within the bone marrow, promoting the release of mature neutrophils into peripheral circulation. In addition to increasing neutrophil numbers, G-CSF enhances several critical neutrophil functions, including chemotaxis, antibody-dependent cellular cytotoxicity (ADCC), immunoglobulin A (IgA)-mediated phagocytosis, and superoxide production. These enhanced functions significantly contribute to improved microbial clearance and a more robust immune response in treated subjects (Roberts,

2005) [18].

Conclusion

The present study evaluated the clinical and haematological alterations in dogs affected with parvoviral enteritis and assessed the therapeutic efficacy of filgrastim as an adjunct to standard supportive treatment. All dogs exhibited severe clinical signs including inappetence, vomiting, diarrhoea, and dehydration, along with notable leukopenia and neutropenia at presentation. Standard supportive therapy led to gradual clinical improvement and restoration of haematological parameters; however, the addition of filgrastim resulted in a significantly faster increase in total leukocyte and neutrophil counts. This accelerated haematological recovery was associated with a markedly shorter recovery period compared to dogs receiving standard treatment alone. Biochemical parameters showed progressive improvement across all groups during the treatment period. The findings indicate that filgrastim is a safe and effective adjunctive therapy that enhances immune recovery and reduces the duration of illness in dogs with parvoviral enteritis. This study suggests that granulocyte colony-stimulating factor therapy can be beneficial in the management of canine parvoviral enteritis, particularly in cases presenting with severe neutropenia. Further studies with larger sample sizes are warranted to confirm these observations and to establish optimal treatment protocols.

Conflict of interests

The authors declare that they have no competing interests.

Data availability statement

Data set generated during research will be available from the corresponding author on reasonable request.

Ethical statement

The Institutional Animal Ethics Committee (IAEC) following the guidelines of Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA) approved the research work on the animals.

Owners consent

Prior informed consent was obtained from all dog owners before initiation of the study. Owners were thoroughly informed about the objectives and nature of the study, the diagnostic procedures involved, and the intended use of clinical data for research purposes.

Author's contribution

All authors made substantial contributions to the conception and design of the study. Clinical examination, data collection, and diagnostic evaluations were conducted by the primary author with assistance from the co-authors. Data analysis and interpretation were performed by the corresponding author with the help of co-authors. The manuscript was prepared by the primary author and was critically reviewed, revised, and approved by all co-authors. All authors have read and approved the final version of the manuscript.

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