



Predictors of the Duration of Management and Clinical Outcomes in Dogs with Acute Gastroenteritis

Felix Kundu Shima^{1*}, Tersoo Uba², Thaddaeus Ternenge Apaa³, Matthew Terzungwe Tion³, Temidayo Olutayo Omobowale¹ and Helen Oyebukola Nottidge¹

¹Department of Veterinary Medicine, University of Ibadan, Nigeria

²Department of Statistics, Joseph Sarwuan Tarka University, Nigeria

³Department of Veterinary Medicine, Joseph Sarwuan Tarka University, Nigeria

*Corresponding author: Felix Kundu Shima, Department of Veterinary Medicine, University of Ibadan, Ibadan, Nigeria

Received: 📅 February 02, 2022

Published: 📅 February 14, 2022

Abstract

Canine gastroenteritis represents one of the major presenting disorders in canine practice with challenges in its diagnosis and management. Its prognosis is reliant on the aetiology, severity of gastroenteritis, and response to treatment. Knowledge of factors influencing the course of this disorder is essential. This study evaluates prognosticators that can be utilised in predicting the clinical outcome and duration of management for dogs presenting with gastroenteritis. To achieve this, the clinical pathology profile of 104 dogs with gastroenteritis managed on outpatient was assayed using standard procedures. Data analysis was conducted using descriptive, Pearson's correlation, and logistic regression model at $\alpha^{0.05}$ significance level. Haematological changes noted before treatment were anaemia, monocytopenia, lymphocytosis, leukopenia, neutropenia, thrombocytopenia, pancytopenia, hyperfibrinogenaemia, and hypofibrinogenaemia. Biochemical changes were azotaemia, mild hyperglycaemia, hyperglobulinaemia, hyponatraemia, elevated albumin/globulin ratio, alanine aminotransferase, and alkaline phosphatase, hypercreatininaemia, hyperproteinaemia, hyperalbuminaemia, hypoalbuminaemia, hypokalaemia, hypercalcaemia, hypochloraemia, decreased aspartate aminotransferase and hypoproteinaemia. Reduction in the three blood cellular components (*pancytopenia*: $OR = 0.2$) or leukopenia ($OR = 3.5$) prolonged duration of management by a day above the median duration of five days. Similarly, leukopenia ($OR = 0.1$), hypoalbuminaemia ($OR = 7.1$), and colic ($OR = 0.01$) were associated with a poor prognosis. Leukopenia was the only clinicopathologic abnormality that influenced both the duration of management and clinical outcome. The progression of gastroenteritis may not be determined by its cause but the presence of colic, hypoalbuminaemia (*serum albumin conc.* $\leq 20\text{mg/dL}$), leukopenia ($WBC \leq 4,500\text{ cells/L}$), and pancytopenia before treatment may predict clinical outcome and the duration of management for dogs presenting with gastroenteritis.

Keywords: Canine Practice; Sampling; Logistic Regression; Biomarkers; Prognosis; Outpatient.

Introduction

The functions of the digestive system include food digestion and absorption, toxins neutralisation and elimination of wastes products from the body. Gastroenteritis is one of the important disorders of the gastrointestinal tract which has caused high morbidity and several deaths of dogs in Nigeria [1-3]. Gastroenteritis represents one of the major presenting disorders

in canine practice with challenges in its diagnosis and management [4]. The aetiologies are multifactorial and can be viral, bacterial, or protozoan, non-infectious, and systemic non-gastrointestinal diseases. Its management involves symptomatic treatment, rehydration, or prevention of dehydration, and/or management of secondary bacterial infections. Clinical pathological assessment

is useful in establishing differential diagnoses, assessing response to treatment, and setting prognosis [5,6]. Good knowledge of the several biomarkers and predictors of the progression of a disease is relevant for establishing appropriate treatment and identification of patients that are critically ill [7]. Biomarkers are the several numbers of objective normal or abnormal findings observed from a patient under physical or clinical examination, indicating functional physiology or pathological status. Meaning clinicians must be able to repeatedly measure such clinical findings correctly and consistently [8]. There are reports of marked alterations in the clinicopathologic indices of patients with gastroenteritis, with electrolyte imbalance and dehydration as the most common [5,9,10]. Expanding knowledge and effective prognostication can be beneficial in managing this disorder. This study was conducted to assess clinical pathologic profiles of Nigerian dogs with gastroenteritis, and their usefulness in diagnosis and prognostication. We hypothesized that regardless of the aetiology involved, changes in clinical pathology profiles of dogs with gastroenteritis at presentation are useful in predicting outcome and duration of management.

Ethical Considerations

This study received approval from the University of Ibadan Animal Care and Use Research Ethics Committee (protocol number UI – ACUREC / App / 03 / 2017 / 007) and consent from the dog owners and the management of the veterinary clinics sampled.

Materials and Methods

Sampling

A total of 104 client-owned dogs were sampled from five selected veterinary clinics in Nigeria, using a non-probability haphazard sampling method. Only cases with gastroenteritis that have not been treated before presentation, and were screened for Canine Parvovirus (CPV), Coronavirus (CCoV), *Giardia*, intestinal parasites were eligible for inclusion in the study. Demographic and qualitative data collected were breed, age, sex, husbandry practice, anaemia, anorexia, fever, depression/lethargy, dehydration, abdominal pains, body condition score, worming history, vaccination history, co-infection, diagnosis, faecal consistency (formed, watery, bloody, mucoid), frequency/duration of diarrhoea, frequency/duration of vomiting, the Duration of Management (DOM) and outcome (Death, Euthanasia, or Recovery). The DOM was defined as a difference in the number of days between first presentation and recovery or discharge. Qualitative data, blood samples for haematology and biochemical tests, as well as faecal samples, were collected once from each patient at initial presentation. The dogs were managed as out-patients and treatment were given based on the assessed need of each patient.

Laboratory Analyses

The dogs were screened first for CCoV, CPV, and *Giardia* using on-the-spot immunoassay kits (SensPERT[®], VetAll Laboratories, Korea). Coprological techniques for laboratory screening of endoparasites were done as described by Urquhart et al. [11]. Blood samples for haematology and serum chemistry were analysed using commercial kits and standard procedures (Randox Laboratories Ltd, UK).

Data Handling and Statistical Analysis

The dogs ($N=104$) were grouped based on responses to treatment: survivors ($n=70$) and non-survivors ($n=34$). To ascertain the strength of the associations and predictive ability of the covariates influencing the prognosis and the DOM (below or above the median duration), biochemical and haematological indices were coded as being either within or outside the normal reference limits. Also, qualitative variables were classified as either present or absent, vaccinated or nonvaccinated, dewormed or non-dewormed, viral, or other diseases, etc. The hematologic and biochemical variables were presented as a mean \pm standard error of the mean. Descriptive statistics, Pearson's correlation, and logistic regression were used in analysing the data. Pearson's correlation was performed and variables demonstrating significant correlation with DOM and clinical outcomes were selected and further analysed by logistic regression model using the enter method. Hosmer-Lemeshow χ^2 test, the omnibus test of model coefficients, and Nagelkerke's pseudo- R^2 were performed to ensure the final model was well fitted. All Statistical analyses were performed with SPSS (v20, IBM) for Windows and evaluated at $p < 0.05$ significance level.

Results

Table 1 summarises the characteristics of the sampled dogs. The patients ($N=104$) studied comprised of Alsatiens ($n_1=29$), Boerboels ($n_2=20$), Rottweilers ($n_3=20$), Crossbreeds ($n_4=18$), Caucasia ($n_5=11$), and other least represented dog breeds ($n_6=6$). The dogs aged 7.7 months ($95\% \text{ CI} = 5.7-9.7$). Eighty dogs were ≤ 6 months old and were mostly males (57). The dogs had fair (39) and emaciated (42) body condition scores. Ninety-seven were restricted dogs, while 7 strayed. Gastroenteritis was more prevalent in restricted dogs (97 cases) than stray dogs (7 cases). More than 50% of the dogs (56) were dewormed within the last 3 months before the illness. Diseases detected were majorly viral (93) and non-viral (11). Two-third (70) of the patients survived, whereas one-third (34) died. Two-third (70) received at least a dose of the DHLPP vaccine, while one-third was unvaccinated. Clinicopathologic changes and variables influencing DOM and outcomes are presented in Tables 2–6.

Table 1: Characteristics of dogs with acute gastroenteritis.

Variables	Category	Total	
		Frequency (n)	Percentage (%)
Breed	Alsatian	29	27.9
	Boerboel	20	19.2
	Rottweiler	20	19.2
	Crosses	18	17.3
	Caucasian	11	10.6
	Others ^c	6	5.6
Age ^a	0–6 months	80	76.9
	7–11 months	17	16.3
	≥12 months	7	6.7
Sex	Male	57	54.8
	Female	47	45.2
Body score ^b	Good	23	22.1
	Fair	39	37.5
	Emaciated	42	40.4
Lifestyle	Restricted	97	93.3
	Strays	7	6.7
Worming history	Not dewormed	48	46.2
	Dewormed	56	53.8
DHLPP vaccination history	Unvaccinated	34	32.7
	Vaccinated	70	67.3
Diagnosis	Viral diseases	93	89.4
	Helminthosis	5	4.8
	Hepatic diseases	1	0.9
	Colitis/gastritis	3	2.9
	Undetermined	2	1.9
Treatment outcome	Survived	70	67.3
	Died	34	32.7
	Grand total (N)	104	100

^aMean age of the dogs (7.7 months, median = 4 months, CI = 5.7–9.7).

^bMean weight of the dogs (13.5kg, CI = 11.9–15.2); Average number of dogs per household (2.3±0.3; CI = 1.9–2.7).

^cOthers (least represented breeds = Great Dane, Bullmastiff, Lhasa Apso, Pitbull, Doberman, and Indigenous).

Table 2: Clinical signs recorded in dogs with acute gastroenteritis.

Clinical signs	Sub-category	Frequency (n)	Percentage (%)
Anorexia	-	92	88.5
Abdominal pain	-	10	9.6
Dehydration	-	68	65.4
Depression	-	60	57.7
Diarrhoea		100	96.1
	Bloody	69	66.3
	Watery or mucoid	31	29.8
Lethargy	-	34	32.7
Fever	-	31	29.8
Mucosa pallor	-	48	46.2

Vomiting	-	75	72.1
Faecal consistency	Watery without solid	89	85.6
	Sloppy and mushy	7	6.7
	Soft stool	7	6.7
Tick infestation	-	25	24.0

The mean rectal temperature (39.4°C, CI = 39.2-39.6°C); Mean respiratory rate (44.6 breaths/min, CI = 36.9-52.4); Mean heart rate (135.7±6.5 beats/min, CI = 123-148.5); Mean diarrhoea episode (3.1±0.3, median = 2; CI = 2.6-3.6 times); Mean vomiting episodes (2.4±0.3, median = 1, CI = 3.0-1.8 times); Mean duration of diarrhoea (3.7±0.3, median = 3, CI = 3.2-4.2 days); Mean duration of clinic signs before presentation (3.3±0.3, median = 3, CI = 2.8-3.9 days); mean duration of management (5.3±0.3, median 5, CI = 4.8-5.9).

Table 3: Haematologic changes in dogs with acute gastroenteritis.

Abnormality observed	No. of patients n (%)	Observed range (mean)	^b Reference range
Anaemia (PCV, %)	49 (47.1)	26-28.9	35 - 57
Haemoconcentration (PCV, %)	1 (0.9)	---	35 - 57
Low Hb (g/dL)	41 (39.4)	8.8-10	11.9 - 18.9
Low RBC (μL)	25 (24.0)	3.8-4.5	4.95 - 7.87
High RBC (μL)	9 (8.6)	8.5-8.8	4.95 - 7.87
Low MCV (fL)	60 (57.7)	60.1-61.3	66 - 77
Low MCH (pg)	59 (56.7)	19.5-20	21 - 26.2
Low MCHC (g/dL)	13 (12.5)	28.8-31.2	32 - 36.3
Leukopaenia (μL)	32 (30.8)	3.4-4	5 - 14.1
Leucocytosis (μL)	10 (9.6)	15.9-28.8	5 - 14.1
Thrombocytopaenia (μL)	49 (47.1)	122.7-150.5	211 - 621
Thrombocytosis (μL)	1 (0.9)	---	211 - 621
Neutropaenia (%)	28 (26.9)	40.4-47.8	58 - 85
Neutrophilia (%)	8 (7.7)	86.4-91.4	58 - 85
Lymphopenia (%)	7 (6.7)	4.5-6.1	8 - 21
Lymphocytosis (%)	55 (52.9)	34.2-40.6	8 - 21
Monocytopenia (%)	15 (14.4)	0.2-0.7	2 - 10
Monocytosis (%)	3 (2.8)	10-14	2 - 10
Eosinophilia (%)	1 (0.9)	---	0 - 9
Pancytopenia ^a	29 (27.9)	PCV 26.4-31.1; WBC 4.3-5.5 PLT 109.9-143.9	35 - 57 5.0 - 14.1 211 - 621
Hypofibrinogenemia (mg/dL)	16 (15.4)	---	150 - 300
Hyperfibrinogenaemia(mg/dL)	20 (19.2)	446.4-513.7	150 - 300

^aPancytopenia was defined as the combination of low haematocrit (PCV, <37% for dogs older than five months or <30% for dogs younger than five months), white blood cell counts (WBC, <6×10³/L) and platelets (<200×10³/L) counts [12].

^bReference range [13].

Table 4: Serum biochemical changes in dogs with acute gastroenteritis.

Abnormality	No. of patients (n, %)	Observed range (mean)	^a Reference range
Hypoproteinaemia (g/dL)	9 (8.7)	4.6-5.1	5.4 - 7.5
Hyperproteinaemia (g/dL)	35 (33.7)	8-8.3	5.4 - 7.5
Hypoalbuminaemia (g/dL)	39 (37.5)	1.6-1.8	2.3 - 3.1
Hyperalbuminemia (g/dL)	17 (16.3)	3.5-3.8	2.3 - 3.1
Hypoglobulinaemia (g/dL)	2 (1.9)	2.3-2.7	2.7 - 4.4

Hyperglobulinaemia (g/dL)	69 (66.3)	4.9–5.0	2.7 – 4.4
Raised A/G ratio	58 (55.8)	2.3–2.7	0.8 – 1.7
Hyperglycaemia (mg/dL)	29 (27.9)	137–139.8	76 – 119
Hypercreatinaemia (mg/dL)	47 (45.2)	2.1–2.2	0.5 – 1.7
Azotaemia (mg/dL)	78 (75)	37.4–39.8	8 – 28
Raised Urea/Creatinine ratio	6 (5.8)	26.4–34.7	4 – 27
Raised ALP (u/L)	57 (54.8)	122.8–126.2	1 – 114
Raised ALT (u/L)	75 (72.1)	118.4–121.4	10 – 109
Low AST (u/L)	10 (9.6)	11.4–12	13 – 15
Raised AST (u/L)	2 (1.9)	---	13 – 15
Hypokalaemia (mEq/L)	27 (26.0)	3.4–3.6	3.9 – 5.7
Hyperkalaemia (mEq/L)	1(0.9)	---	3.9 – 5.7
Hyponatraemia (mEq/L)	64 (61.5)	134.5–136.7	142 – 152
Hypercalcaemia (mEq/L)	26 (25.0)	12.5–12.8	9.1 – 11.7
Low Na/K ratio	1 (0.9)	---	25 – 40
Raised Na/K ratio	8 (7.7)	42.4–43.6	25 – 40
Hypochloraemia (mEq/L)	19 (18.3)	104.7–107.4	110 – 124
Hyperchloremia (mEq/L)	4 (3.8)	124.8–126.7	110 – 124

Tp = total proteins; Alb = albumin; Glb = globulin; A/G ratio = albumin to globulin ratio; BUN = blood urea nitrogen; ALP = alkaline phosphatase, ALT = alanine aminotransferase; AST = aspartate transaminase; Crt. = creatinine; aReference range [13].

Table 5: Correlations of some studied variables with outcome and duration of management.

Variables	Correlation (R ²)	p-value
Clinical outcome		
White blood cells	0.44	<0.001 ^a
Albumin	-0.28	0.008 ^a
Anaemia	0.20	0.04 ^b
Mucosa pallor	0.22	0.026 ^b
Duration of management	-0.36	<0.001 ^a
Depression	0.22	0.023 ^b
Lethargy	0.21	0.03 ^b
Abdominal pain	0.33	0.001 ^a
Duration of management		
Packed cell volume	0.21	0.033 ^b
Haemoglobin	0.24	0.039 ^b
White blood cells	-0.24	0.015 ^b
Pancytopenia	0.32	0.001 ^a
Total proteins	-0.26	0.015 ^b
Alkaline phosphatase	0.21	0.05 ^b
Sex	-0.23	0.017 ^b
Lifestyle	-0.20	0.038 ^b
Duration of diarrhoea	0.26	0.009 ^a

^aCorrelation was significant at the 0.01 level.

^bCorrelation was significant at the 0.05 level, N=104.

Table 6: Risk of poor prognosis and prolonged duration of management in the distinct groups compared to normal (OR =1).

Characteristics	OR (95%CI)	Albumin	WBC	Colic	Pancytopenia
Clinical outcome	Crude	7.1 (1.8-28.6)	0.1 (0.02-0.4)	0.01 (0.0-0.1)	-
	p-value	0.006	0.002	0.001	-
Duration of management	Crude	-	3.5 (1.3-9.1)	-	0.2 (0.1-0.5)
	p-value		0.01		0.002

Note: CI = confidence interval. Changes in albumin concentration had increased likelihood with 7.1 times the odds of death for patients with hypoalbuminemia than patients without. Changes in total WBC and presence of colic had decreased likelihood and are 0.1 and 0.01 times respectively the odds of death for patients with leukopenia and colic than those without, all things being equal. Changes in WBC had increased likelihood with 3.5 times the odds of death for patients with leukopenia than patients without. The presence of pancytopenia had a decreased likelihood and has 0.2 times the odds of death for patients, all things being equal.

Discussion

This study assessed the importance of history taking, clinical examination, and clinical pathology profiles of dogs before treatment. This was to enable us to predict the duration of management and outcome in cases of canine gastroenteritis examined. Canine gastroenteritis was more prevalent in puppies below seven months, validating the report of Wells and Hepper [14]. Puppies are immunologically incompetent and maternally-derived antibodies wane early in life making them more vulnerable to infections [15]. The common clinical findings presented in Table 2 corroborate the reports of Castro, et al. [16]. Gastrointestinal inflammatory processes trigger anorexia, pyrexia, and weight loss by the release of inflammatory endogenous mediators, interleukin-1, and cachectin [17,18]. Haematologic changes recorded in Table 3 tally with findings from previous reports [6,19,20]. The microcytic hypochromic anaemia in this report may be linked to iron deficiency, following intestinal blood loss and inflammations from haemorrhagic gastroenteritis [21,22]. Neutrophilia may be due to immune system stimulation to control opportunistic bacteria during the onset of the disease. The later stages of viral infections are characterised by severe neutropaenia due to the destruction of immune cells and bacterial colonisation of the gut.

Biochemical responses recorded in the dog patients revealed that about 75% of them were azotaemic; comparable to existing findings [23]. This is attributable to dehydrating gastroenteritis and pre-renal uraemia with a reduction in glomeruli filtration rate or catabolic breakdown of tissues in pyretic animals [24]. The sampled dogs had a temperature range of 39.2–39.6°C, suggestive of pyrexia due to bacterial infections. The high rates of hyperglobulinaemia, hypoalbuminaemia, increased A:G ratio, hyperalbuminemia, and hypoproteinaemia observed corroborated previous reports [25,26]. Changes in total proteins were related to a marked reduction in dietary intake, malabsorption, and protein-losing enteropathy [25,26] hepatic synthesis of acute-phase proteins by damaged GI tissue and inflammation [27], destruction of the intestinal villi by infectious pathogens or systemic inflammatory response syndrome (SIRS)-mediated increased vascular permeability [28]. Increased A:G ratio can be equated to an increase in albumin with a relative decrease in globulin concentration. The hyperglycaemia reported

here may reflect dehydration, stress, or ketosis that interfered with glucose utilization by the patients. This hyperglycaemia is transient and resolved following rehydration. This has been described in children suffering from severe gastroenteritis [29] and dogs with viral enteritis. Elevation in liver enzymes (ALP, ALT, BUN, and creatinine) concurs with previous reports [9,30]. These changes could be triggered by dehydration, hepatic hypoxia secondary to severe hypovolaemia or absorption of toxic wastes [30,31] or the young age of the sampled dogs [32].

Low levels of AST in 9.6% of the dogs sampled is unexplainable. However, malabsorption and malfunctioning or damaged liver prevented the release of enough quantities into circulation. The dogs sampled had mostly hyponatraemia, hypokalaemia, hypochloroemia, raised Na: K ratio, and hypercalcaemia, corroborating the findings of previous studies. Disturbances in these electrolytes have been linked to vomiting and diarrhoea or failure of the colon to conserve potassium by the patients [33]. Metabolic, systemic, and parasitic diseases such as hypoadrenocorticism, pancreatic disease, pyometra, renal diseases are the established causes of low Na: K ratio in dogs [34]. Hypercalcaemia may be relative to hyperalbuminemia or dehydrating gastroenteritis. Biochemical changes such as hypoproteinaemia, hypoalbuminaemia, hypoglycaemia, and hyperglycaemia are associated with interactions between multiple factors such as malnutrition, septicaemia, stress-induced activation of catecholamines, hypocalcaemia, hypokalaemia, hyponatraemia, hypochloroemia, hypomagnesaemia, azotaemia, liver damage induced by hypoperfusion and SIRS [6,35,36].

In the correlation analysis, total leukocyte counts, mucosae pallor, depression, lethargy, abdominal pain, and anaemia correlated with clinical outcome (Table 5). However, only changes in albumin, total WBC count, and colic had the strongest tendencies to influence prognosis (Table 6). The odds of death for dogs with hypoalbuminaemia (serum albumin conc. ≤ 20 mg/dL), leukopaenia ($WBC \leq 4,500$ cells/L), and presence of colic were 7.1, 0.1, and 0.01 times, respectively the odds of death for patients without these changes. Leukopaenia and hypoalbuminaemia reported here tally with similar studies. The finding of leukopaenia as a biomarker of poor prognosis corroborates existing published reports [37,38].

Some researchers argued that neutrophils are more prognostic than leukocytes, when there is severe neutropaenia leukopaenia ensues, hence leukopaenia alone ought not to form the sole criterion of prognosis [37,39]. In contrast to that claim, severe neutropaenia was not found to be prognostic [40,41]. Contrary to the finding reported here and that of Mason et al. [39], Glickman, et al. [42] also reported that leukopaenia is not a biomarker of poor prognosis. The finding of hypoalbuminaemia as a biomarker of poor prognosis in this study concurs with the report of Boag [43]. Association of colic with poor prognosis may suggest the severity of the disease, intussusceptions, inflammation of the gut mucosae, or secondary infections. Besides the findings reported here,

lymphopenia, vomiting, depression, body weight, changes in blood glucose, sodium, and AST concentrations [44] and how severe the disease is [45] determine the clinical outcome. Variables that correlated with DOM in this investigation included changes in PCV, WBC, Hb, pancytopenia, total proteins, ALP, sex of the dog, lifestyle, and duration of diarrhoea (Table 7). The odds of pancytopenia and leukopenia to prolong DOM in dogs with gastroenteritis by at least a day above the median five days were 0.2 and 3.5 times, respectively. In contrast, vomiting, depression, lymphopaenia, and hypoalbuminaemia were reported to prolong the duration of hospitalisation [6,46].

Table 7: Treatment options used in the management of the canine gastroenteritis cases studied indicating the drug used, dosage, frequency, route of administration, and percentage of patients who received a particular treatment.

Prescriptions	Dose, frequency, and routes of administration	Notes
Antibacterials		
Metronidazole ^a	7.5mg/kg/daily, IV; Max.65mg/kg/day	Used in >80% of the cases
Gentamicin ^b	6mg/kg/day, IV	Used in >80% of the cases
Amoxicillin ^c	22mg/kg/day, IV	Used in >80% of the cases
Oxytetracycline 5%	1ml/5kg/day, IV, IM	Used in 19% of the cases
Sulphadimidine (333mg/100ml)	0.6mL/kg first day, subsequently 0.3mL daily for 3-4 days.	Used in 8% of the cases
Enrofloxacin	5-10mg/kg/day, IV	Used in 4% of the cases
Phthalylsulfathiazole (Thalazole [®])	1tablet 3 times daily for at least 2 days, PO	Used in 3% of the cases
Antiemetics		
Metoclopramide ^d	1-2mg/kg/day IV, IM	Used in 50% of the cases
Antihemorrhagics		
Dicynone [Etamsylate 125mg/ml]	0.5-1mL stat, IM	Used in 20% of the cases
Vitamin K1	0.25-5mg/kg stat, IM	Used in 3% of the cases
Anthelmintics		
Ivermectin	0.2mg/kg stat, SC	Used in 14% of the cases
*Prazisam [®] , Caniverm [®]	1 tablet/10kg stat, PO	Used in 9% of the cases
Pyrantel pamoate [62.5 mg Tablet]	5-10mg/kg stat, PO	Used in 4% of the cases
Fluid therapy^e		
5% Dextrose saline	44mL/kg/day, IV	Administered to all case
Lactated Ringer's solution	65mL/kg/day, IV	Administered to all case
Gastroprotectants		
Cimetidine	5-10mL/day, PO	Used in 3% of the cases
Immunomodulators		
Dexamethasone	0.2mg/kg/day, IM	Used in 2% of the cases
Prednisolone	2mg/kg/day, PO	Used in 1% of the cases
Vitamins and Minerals^f		
B-complex	1-5mL/day, IM	Used in 90% of the cases
Multivitamins	1-5mL/day, IM	Used in 10% of the cases

*NOTE-Prazisam[®]: Fixed-dose formulary of anthelmintics containing praziquantel 50mg, pyrantel pamoate 144mg, and fenbendazole 500mg.

Caniverm[®]: Praziquantel 50mg, pyrantel pamoate 144mg, and fenbendazole 150mg. The dogs were prescribed ^{a, b, c, d, e, f} extensively. The treatment options were decided by the clinicians based on the need of the individual case. The median duration of treatment from reporting to discharge was 5 days.

Leukopaenia was the only clinical pathology at admission that significantly influenced both clinical outcomes and the DOM as opposed to previous findings [6,42]. Also, pancytopenia significantly increased the DOM as opposed to reports that it influences poor clinical outcomes in many infectious diseases [47]. The pancytopenia dogs studied improved with treatment, making pancytopenia a nonsignificant prognosticator of poor outcomes. Ultimately, Goddard, et al. reported that an accurate prognosis may be obtained 24 hours postadmission. But this report showed that changes in total leukocyte counts (specifically leukopaenia), reduction in the three blood cellular components (pancytopenia), albumin (particularly hypoalbuminaemia) and presence of colic at presentation are prognostic for poor outcomes and prolonged DOM in dogs with gastroenteritis. Interestingly, the type of disease is not a good predictor of clinical outcome and DOM in this study. These findings may assist clinicians in recognizing canine gastroenteritis candidates with substantial risk, and rapid selection of appropriate treatment and intensive care measures.

Acknowledgements

Colleagues, management of the veterinary clinics, as well as owners of the dogs are duly acknowledged for the consent and access granted to their facilities, animals, and samples collected.

References

- Shima FK, Mosugu JI, Apaa TT (2014) Causes of mortality in dogs in and around Effurun/Warri Municipality of Delta State, Nigeria. *Bulletin of Animal Health and Production in Africa* 62: 387-396.
- Shima FK, Tion MT, Mosugu JI, Apaa TT (2015) Retrospective study of disease incidence and other clinical conditions diagnosed in owned dogs in Delta State, Nigeria, *Journal of Advanced Veterinary and Animal Research* 2: 435-449.
- Shima FK, Omotosho OO, Apaa TT, Omobowale TO, Nottidge HO (2021) A retrospective study of the prevalence of gastroenteritis in dogs attending some veterinary clinics in Nigeria. *Rev Vét Clin* 56: 170-176.
- Tams TR (2003) Gastrointestinal symptoms. *Handbook of Small Animal Gastroenterology*. TR Tams Ed. 2nd ed. Elsevier, USA.
- Goddard A, Leisewitz AL, Christopher MM, Duncan NM, Becker PJ (2008) Prognostic usefulness of blood leukocyte changes in canine parvoviral enteritis. *Journal of Veterinary Internal Medicine* 22: 309-316.
- Kalli I, Leontides LS, Mylonakis ME, Katerina Adamama-Moraitou, Timoleon Rallis, et al. (2010) Factors affecting the occurrence, duration of hospitalisation and final outcome in canine parvovirus infection. *Research in Veterinary Science* 89: 174-178.
- Schoeman JP, Goddard A, Leisewitz AL (2013) Biomarkers in canine parvovirus enteritis. *New Zealand Veterinary Journal* 61: 217-222.
- Strimbu K, Tavel JA (2010) What are biomarkers? *Current Opinion in HIV and AIDS* 5.6: 463-466.
- Salem NY (2014) Canine viral diarrhoea: clinical, haematologic and biochemical alterations with particular reference to in-clinic rapid diagnosis. *Global Veterinaria* 13(3): 302-307
- Kumar A, Kumar RK (2017) Clinico-hematobiochemical changes in parvoviral infection in dog. *International Journal of Science, Environment and Technology* 6: 2837-2841.
- Urquhart GM, Duncan JL, Armour J, Dunn AM, Jennings FW (1996) *Veterinary Parasitology*. 2nd edn. Blackwell Science Ltd, Oxford, USA pp. 53-73.
- Weiss DJ, Evanson OA, Sykes J (1999) A retrospective study of canine pancytopenia. *Veterinary Clinical Pathology* 28: 83-88.
- Merck (2016) Reference guides. *The Merck Veterinary Manual*.
- Wells DL, Hepper PG (1999) Prevalence of disease in dogs purchased from an animal rescue shelter. *Veterinary Records* 144: 35-38.
- Jarret O, Ramsey I (2001) Vaccination. *Manual of canine and feline infectious diseases*. Ramsey I, Gloucester TB (Eds), British Small Animal Veterinary Association, The UK, pp. 41-50.
- Castro TX, Miranda SC, Labarthe NV, Silva LE, Cubel Garcia RCN (2007) Clinical and epidemiological aspects of canine parvovirus (CPV) Enteritis in the State of Rio de Janeiro: 1995-2004. *Arquivo Brasileiro de Medicina Veterinariae Zootecnia* 59: 333-339.
- Goddard A, Leisewitz AL (2010) Canine parvovirus. *Veterinary Clinics of North America: Small Animal Practice* 40: 1041-1053.
- Sharp CR, Declue AE, Haak CK, Honaker AR, Reinero CR (2010) Evaluation of the anti-endotoxin effects of polymyxin B in a feline model of endotoxaemia. *Journal of Feline Medicine and Surgery* 12: 278-285.
- Haligur M, Ozmen O, Sezer K, Sahinduran S (2009) Clinical, pathological and immunohistochemical findings in diarrheic dogs and evaluation of canine parvoviral and coronaviral enteritis. *Journal of Animal and Veterinary Advances* 8: 720-725
- Decaro N, Buonavoglia C (2012) Canine parvovirus - a review of epidemiological and diagnostic aspects, with emphasis on type 2c. *Veterinary Microbiology* 155: 1-12.
- Naigamwalla DZ, Webb JA, Giger U (2012) Iron deficiency anaemia. *Canadian Veterinary Journal* 53: 250-256.
- Torrente C, Manzanilla EG, Bosch L, Fresno L, Rivera Del Alamo M, et al. (2015) Plasma iron, C-reactive protein, albumin, and plasma fibrinogen concentrations in dogs with systemic inflammatory response syndrome. *Journal of Veterinary Emergency and Critical Care* 25: 611-619.
- Bhat AA, Wadhwa DR, Singh SP, Singh I (2013) Haematological and biochemical analysis in canine enteritis. *Veterinary World* 6: 380-383.
- Shinde SS, Rajguru DN, Anantwar LG, Mohd S, Ambore BN (2000) Clinicopathology and therapeutic management of canine gastroenteritis. *The blue cross book* 15: 26-29.
- Dossin O, Lavour R (2011) Protein-losing enteropathies in dogs. *Veterinary Clinics of Small Animal* 41: 399-418.
- Craven M, Caroline SM, Kenneth WS (2011) Granulomatous colitis of Boxer dogs. *Veterinary Clinics of Small Animal* 41: 433-445.
- Broek AH (1990) Serum protein electrophoresis in canine parvovirus enteritis. *British Veterinary Journal* 146: 255-259.
- Mazzaferro EM, Rudloff E, Kirby R (2002) The role of albumin replacement in the critically ill veterinary patient. *Journal of Veterinary Emergency Critical Care* 12: 113-124.
- Seth A, Aneja S (1995) Hyperglycemia in malnourished children with dehydrating gastroenteritis. *Indian Journal of Paediatrics* 62: 353-355.
- Shah SA, Sood NK, Wani N, Gupta K, Singh A (2013). Haemato-biochemical changes in canine parvoviral infection. *Indian Journal of Veterinary Pathology* 37: 131-133.
- Berghoff N, Steiner JM (2011) Laboratory tests for the diagnosis and management of chronic canine and feline enteropathies. *Veterinary Clinics of Small Animal* 41: 311-328.

32. Macintire DK, Smith-Carr S (1997) Canine parvovirus. Part II. Clinical signs, diagnosis, and treatment. *Compendium Continuing Education Practice for Veterinarians* 19: 291-302
33. Ettinger JS, Feldman EC (2010) *Textbook of Veterinary Internal Medicine: Diseases of dog and cat*. WB Saunders Company, Philadelphia, USA pp. 1310-1408.
34. Roth L, Tyler RD (1999) Evaluation of low sodium: potassium ratios in dogs. *Journal of Veterinary Diagnostic Investigation* 11: 60-64.
35. Li R, Humm KR (2015) Canine parvovirus infection. In: Silverstein DC, Hoper K (Eds). *Small Animal Critical Care Medicine*. 2nd Ed, St Louis, MO, USA Elsevier pp. 509-513.
36. Mylonakis ME, Kalli I, Rallis TS (2016) Canine parvoviral enteritis: an update on the clinical diagnosis, treatment, and prevention. *Veterinary Medicine: Research and Reports* 7: 91-100.
37. Potgieter LND, Jones JB, Patton CS, Webb-Martin TA (1981) Experimental parvovirus infection in dogs. *Canadian Journal of Comparative Medicine* 45: 212-216.
38. O Sullivan G, Durham PJK, Smith JR, Campbell RSF (1984) Experimentally induced severe canine parvoviral enteritis. *Australian Veterinary Journal* 61: 1-4.
39. Mason MJ, Gillett NA, Muggenburg BA (1987) Clinical, pathological, and epidemiological aspects of canine parvoviral enteritis in an unvaccinated closed beagle colony: 1978-1985. *Journal of the American Animal Hospital Association* 23: 183-192.
40. Brunner C, Swango L (1985) Canine parvovirus infection: Effects on the immune system and factors that predispose to severe disease. *Compendium on Continuing Education for the Practising Veterinarian* 7: 979-989.
41. McCaw DL, Harrington DP, Jones BD (1996) A retrospective study of canine parvovirus gastroenteritis: 89 cases. *Journal of Veterinary Internal Medicine* 10: 157.
42. Glickman LT, Domanski LM, Patronek GJ, Visintainer F (1985) Breed-related risk factors for canine parvovirus enteritis. *Journal of the American Veterinary Medical Association* 187: 589-594.
43. Boag A (2013) Managing parvovirus cases. In: 10th Emergency and Critical Care UK Annual Congress, pp: 200-207.
44. Good JM, Otto CM (2006) Routine biomarkers of sepsis in dogs with canine parvoviral enteritis: a retrospective study (abstract). In: *Proceedings of International Veterinary Emergency and Critical Care Symposium*, pp: 8-9.
45. Mantione NL, Otto CM (2005) Characterisation of the use of antiemetic agents in dogs with parvoviral enteritis treated at a veterinary teaching hospital: 77 cases (1997-2000). *Journal of the American Veterinary Medical Association* 227: 1787-1793.
46. Schoeman JP, Van Schoor M (2008) Admission serum biochemical parameters are not associated with mortality in puppies with parvoviral diarrhoea (abstract). In: *Proceeding's 18th European College of Veterinary Internal Medicine-Companion Animal Congress*, 4th-6th September 2008, Ghent, Belgium, Japn pp. 227-228.
47. Frezoulis PS, Angelidou E, Karnezi D, Oikonomidis IL, Kritsepi-Konstantinou M, Kasabalis D, Melonakos ME (2017) Canine pancytopenia in a Mediterranean region: a retrospective study of 119 cases (2005-2013). *Journal of Small Animal Practice* 58(7): 395-402.

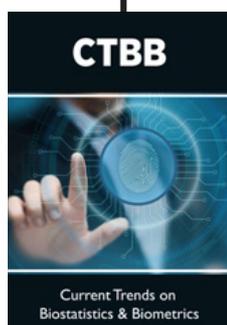


This work is licensed under Creative Commons Attribution 4.0 License

To Submit Your Article Click Here:

[Submit Article](#)

DOI: [10.32474/CTBB.2022.03.000169](https://doi.org/10.32474/CTBB.2022.03.000169)



Current Trends on Biostatistics & Biometrics

Assets of Publishing with us

- Global archiving of articles
- Immediate, unrestricted online access
- Rigorous Peer Review Process
- Authors Retain Copyrights
- Unique DOI for all articles