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Research Article

Clincopathological Findings in Some Cases of Canine Gastroenteritis in Nigeria

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Abstract

Canine gastrointestinal diseases may be characterized by myriads of nonspecific clinicopathologic responses. Adequate knowledge of the clinicopathological spectrum of canine gastrointestinal diseases may provide relevant baseline reference values for diagnosis and prediction of disease outcomes. This study evaluates the clinical haematological and biochemical spectrum of gastroenteritis from 104 dogs consisting of 67.0% survivors and 33.0% non-survivors, using t-test. While 89.4% of dogs evaluated were due to viral enteritis, 10.6% were admitted due to non-viral diseases.The duration between observed clinical signs and presentation to the clinic ranged from 3 to 4days. Clinicopathologic responses recorded at the initial presentation included anaemia, azotaemia, hyperglobulinaemia, hyperglycaemia, hyponatraemia, lymphocytosis, neutrophilia, thrombocytopenia, hypercreatinaemia, elevated serum levels of albumin/globulin ratio, alkaline phosphatase, and alanine aminotransferase. The mean haematological and biochemical indices of survivors compared with non-survivors showed diminutive non-statistically significant differences ($p>0.05$). Nonetheless, the evaluated haematological and biochemical indices provided a direction for initiating treatment and assisted in further diagnoses. A repeat of the haematological and biochemical analysis a few days post-admission may be desirable to establish a prognosis.

Keywords: Clinical pathology; Dog; Diarrhoea; Gastrointestinal disorders; Vomiting

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Introduction

Gastroenteritis is amongst the foremost health challenges in dogs [1]. It is a frequent cause of high morbidity and death in dogs in Nigeria [2, 3]. Gastroenteritis causes a significant loss of body fluid, electrolytes, and occasionally blood [4]. Several aetiological agents are involved in gastroenteritis in small companion animals, such as non-infectious and infectious, gastrointestinal (GI), and systemic non-gastrointestinal causes. These could occur intercurrently [1]. The most frequently reported causes of gastroenteritis in dogs include viral, bacterial, fungal, and parasitic diseases, irritant drugs and toxins, dietary errors, intestinal dysbiosis or bacterial overgrowth, among others [1, 4-7].

The clinical implications of gastroenteritis comprise anorexia, vomiting, and/or diarrhoea, dehydration, weight loss, and electrolyte imbalance [1, 4, 8]. The clinicopathological changes in GI diseases are nonspecific and insufficient for a definitive diagnosis. However, assessment of such changes could be useful in diagnoses, evaluation of dehydration status, provision of baseline clinicopathological reference values for reassessment, and prediction of the disease outcome [1, 4, 9, 10].

In this study, we evaluated the haematologic and biochemical indices of domestic dogs identified as survivors and non-survivors of canine gastroenteritis from selected veterinary clinics in Nigeria.

Materials and Methods

This study was conducted on 104 dogs, comprising 57 males and 47 females, presented with a history of diarrhoea, and/or vomiting to three tertiary veterinary hospitals and two private clinics selected from Abeokuta, Ibadan, Makurdi, and Warri in Nigeria. A non-probability random sampling method was used to sample the dogs. Data were thereafter collected using a Pro-Forma datasheet. Information requested were the breed of dog, sex, age, weight, diagnosis, and clinical outcome following treatment. Rapid in-clinic immunoassay test kits (SensPERT VetAll Laboratories, Kyunggi-Do, Korea) were used to detect antigens to canine coronavirus, parvovirus, and *Giardia* in the faecal samples of the patients. Microscopic identification of helminths was carried out using direct faecal smear, flotation, and sedimentation techniques as described by Urquhart et al. [11] . Blood samples collected from each dog were used to estimate the haematologic and biochemical indices using commercial kits (Randox Laboratories Ltd, UK) following the manufacturer's instructions and standard procedures. Treatment and management of dogs enrolled in this study were carried out by participating veterinarians following clinical evaluation of each dog patient. The data obtained were analysed using Microsoft Excel 2016 software for Windows (Microsoft Inc., USA). The significant difference between the means (haematologic and biochemical indices) of non-survivors and survivors were evaluated using t-test. A two-tailed distribution rule for heteroscedastic data with an assumption that both the survivor and non-survivor groups have unequal variance was used and evaluated at $\alpha_{0.05}$ significance level.

Results

Of the 104 gastroenteritis dogs evaluated, 93 (89.4%) were due to viral enteritis, while 11 (10.6%) were attributed to non-viral diseases as previously published [3]. The major presenting complaints were anorexia (88.5%), diarrhoea (96.1%), and/or vomiting (72.1%). Thirty-four (33.0%) dogs died while 70 (67.0%) survived. Haematologic findings observed at the initial presentation were anaemia, thrombocytopenia, band neutrophilia, and lymphocytosis. Low PCV (34.3±1.1) and band neutrophilia (3.3±0.6) were remarkable haema tologic responses in dogs that survived (Table 1). Also, biochemical responses were increased urea (37±0.7), globulin (4.7±0.1), albumin/globulin ratio (2.2±0.1), serum alkaline phosphatase (118.5±1.1) and alanine aminotransferase (117.3±1), and glucose (138.4±0.7), creatinine (1.9) and low serum sodium (137.8±0.6) concentrations. All the mean biochemical indices of both groups showed only diminutive differences (Table 2).

Parameter (units)	Pre-treatment		Post-treatment		[a]Reference values
	Mean ± SEM	Range (mean)	Non-survivors n = 34 Mean ± SEM	Survivors n = 70 Mean ± SEM	
PCV (%)	35±0.9	33.2–36.9	36.2±1.7	34.3±1.11	35–57
Hb (g/dL)	11.6±0.4	10.9–12.4	11.6±0.7	11.7±0.5	11.9–18.9
RBCs (10^6/µL)	5.8±0.2	5.4–6.15	5.8±0.4	5.8±0.2	5–7.9
MCV (fL)	61.4±0.4	60.7–62.1	60.8±0.6	61.6±0.5	66–77
MCH (pg)	20.1±0.2	19.8–20.4	19.8±0.3	20.3±0.2	21–26.2
MCHC (g/dL)	32.3±0.2	32–32.7	32.1±0.5	32.4±0.2	32–36.3
Platelets (10^3/µL)	197.4±14.8	168.4–226.3	200.3±24.4	195.9±18.7	211–621
WBCs (10^3/µL)	7.9±0.6	6.6–9.1	6.9±1.1	8.3±0.8	5–14.1
Neutrophils (%)	63.9±1.6	60.8–66.9	61.8±3.1	66.9±1.8	58–85
Band neutrophils (%)	3.2±0.5	2.3–4.1	3±0.5	3.3±0.6	0–3
Lymphocytes (%)	27±1.5	24.1–29.9	28.8±2.9	26.2±1.7	8–21
Monocytes (%)	3.4±0.2	2.9–3.9	3.8±0.5	3.2±0.3	2–10
Eosinophils (%)	2.4±0.3	1.7–3	2.8±1	2.18±0.2	0–9
Basophils (%)	0	0	0	0	0–1
Fibrinogen (mg/dL)	288.2±18.2	252.6–323.9	287±26.9	288.9±24	150–300

Table 1: Haematologic profile of dogs (survivors and non-survivors) with acute gastroenteritis

Key: PCV=packed cell volume (haematocrit); Hb=haemoglobin concentration; RBC=Red blood cells; MCV=mean corpuscular volume; MCH=mean corpuscular haemoglobin; MCHC=mean corpuscular haemoglobin concentration; WBC=total white blood cell counts; [a]Normal reference range [12]; Mean duration of sickness that elapsed before presentation (3.3±0.3, median = 3, CI = 2.8–3.9 days). The mean differences between pre-treated and post-treated groups (rows) for studied parameters are not statistically significant (p>0.05).

Parameter (Unit)	Pre-treatment		Post-treatment		[a]Reference values
	Mean ± SEM	Range (mean)	Non-survivors (Mean ± SEM), n=34	Survivors (Mean ± SEM), n=70	
Plasma protein (g/dL)	7.2±0.1	6.9–7.5	7.2±0.2	7.2±0.2	6–7.5
Total protein (g/dL)	7.7±0.1	6.8–7.3	7.2±0.2	7±0.2	5.4–7.5
Albumin (g/dL)	2.4±0.1	2.2–2.6	2.5±0.1	2.4±0.1	2.3–3.1
Globulin (g/dL)	4.7±0.1	4.5–4.8	4.7±0.1	4.6±0.1	2.7–4.4
A/G ratio	2.2±0.1	2–2.3	2±0.1	2.2±0.1	0.8–1.7
Glucose (mg/dL)	138.4±0.7	137–139.8	138.4±0.5	138.4±0.5	76–119
Urea (mg/dL)	37±0.7	35.6–38.4	35.6±1.3	37.7±0.9	8–28
Creatinine (mg/dL)	1.86	1.8–2	1.8±0.1	1.9±0.1	0.5–1.7
Urea/Creatinine ratio	20.4±0.5	19.4–21.4	19.8±0.8	20.8±0.7	4–27
ALP (u/L)	118.5±1.1	116.3–120.6	116.9±1.6	119.3±1.4	1–114
ALT (u/L)	117.3±1	115.3–119.2	117±1.5	117.4±1.3	10–109
AST (u/L)	14±0.1	13.4–13.9	13.5±0.2	13.8±0.1	13–15
Potassium (mEq/L)	4.1±0.1	3.9–4.4	4.2±0.4	4.1±0.1	3.9–5.7
Sodium (mEq/L)	137.8±0.6	136.6–140	137.7±1.1	137.8±0.7	142–152
Chloride (mEq/L)	115.3±0.7	114–116.7	115.2±1.3	115.4±0.8	110–124
Calcium (mg/dL)	11.3±0.1	11–11.5	11.1±0.2	11.4±0.1	9.1–11.7
Na/K ratio	34.5±0.5	33.5–35.5	35.1±1.2	34.2±0.5	25–40

Table 2: Biochemical profile of dogs (survivors and non-survivors) with gastroenteritis

Key: A/G ratio, albumin to globulin ratio; BUN, blood urea nitrogen; ALP, alkaline phosphatase, ALT, alanine aminotransferase; AST, aspartate transaminase, [a]Reference range [12]; Mean duration of sickness that elapsed before presentation(3.3±0.3, median = 3, CI = 2.8–3.9 days). The mean differences between pre-treated and post-treated groups (rows) for studied parametersare not statistically significant (p>0.05)

Discussion

Most of the dogs examined in this study presented with anorexia, diarrhoea, vomiting, dehydration, and depression. The mean duration between the onset of disease and presentation to veterinary clinics was 2.8–3.9 days. Haematologic responses detected include microcytic hypochromic anaemia, band neutrophilia, thrombocytopenia, lymphocytosis, and mild hyperfibrinogenaemia; comparable to earlier reports on canine viral enteritis [9, 10, 13]. The mean haematologic indices between survivors and non-survivors were diminutive with no significant difference (α>0.05), agreeing with the reports of Kalli et al. [14]. Anaemia was less pronounced in non-survivors compared to survivors. This suggests that the survivors initially had regenerative anaemias or iron deficiency anaemia and/or anaemias due to endoparasites that improved with treatment. Gastrointestinal bleeding

due to canine infection with soil-transmitted helminths such as *Ancylostoma caninum* is often associated with microcytic hypochromic anaemia [15,16]. Also, band neutrophilia was more in survivors. This corroborates existing findings that also reported band neutrophilia besides lymphopenia in dogs with gastroenteritis. Neutrophilia regularly follows acute gastroenteritis due to immune stimulation to control opportunistic bacterial infections or a reduction in the other leukocyte cell types as observed in viral enteritis [17-20].

Biochemical responses in the dogs were azotaemia, hyperglycaemia, hyperglobulinaemia, elevated albumin/globulin ratio, hypercreatinaemia, elevated alkaline phosphatase, alanine aminotransferase levels, and hyponatraemia supporting existing findings [13, 21]. In contrast, these findings differ from previous studies where hypoglycaemia, hypoglobulinaemia, and low albumin/globulin ratio were reported in canine viral enteritis [13, 21]. Therefore, these differences in findings could be associated with study design and the presence of hepatobiliary disorder associated with canine parvoviral infection [22] leading to hypoglycaemia, hypoglobulinaemia, and low albumin/globulin ratio in the aforesaid studies. Comparable to the findings of Kalli et al. [14], diminutive deviations in the mean biochemical values between non-survivors and survivors are not statistically significant. The hyperglycaemia recorded in this study is likely to be associated with dehydration in gastroenteritis, frequently reported canine viral enteritis [10] and children [23]. This hyperglycaemia was temporary and was corrected after fluid restoration. Elevated liver enzymes including alkaline phosphatase, alanine aminotransferase, creatinine, and urea levels in cases studied are likely due to dehydration and passage of microbes and endotoxins via portal circulation precipitating reactive hepatopathy [13, 24, 25]. In young animals with viral infections, alkaline phosphatase activity is usually elevated [26, 27]. This also could explain our findings as the mean age of the dogs was 7.7 months, with the majority been positive for viral enteritis. The hyperglobulinaemia and hypoalbuminemia may be likely due to a marked decline in dietary intake, malabsorption, or protein-losing enteropathy [28, 29]. The α-globulins usually increases with the hepatic synthesis of acute-phase proteins, stimulated by leukocyte endogenous mediators following tissue damage or inflammatory processes [30]. Azotaemia in the studied cases agrees favourably with previous reports [21] and could be due to dehydration and fever as the majority were pyretic with a mean rectal temperature of 39.4°C [31].

Hyponatraemia was the only electrolyte response detected in survivors and non-survivors at presentation, conversely, hypokalaemia and hypochloraemia were reported by Bhat et al. [21]. In agreement with our findings, Dhanapalan et al. [32] found no significant changes in serum concentrations of chloride. The mean difference in serum electrolytes levels between non-survivors and survivors were diminutive and not statistically significant ($\alpha>0.05$), corroborating the report of Kalli et al. [14]. The mean serum concentrations of potassium, calcium, chloride and Na/K ratios in both groups were within their respective reference ranges. Patients with gastroenteritis have a combined loss of electrolytes, nutrients, and water. The electrolytes losses are proportional to the severity and frequency of gastroenteritis [33]. Dogs with gastroenteritis presented within four days of clinical disease onset demonstrated arrays of nonspecific clinicopathological changes with the mean values of survivors and non-survivors showing only diminutive differences that are not statistically significant ($\alpha>0.05$). A repeat of the analyses a few days after admission may be needed to establish prognosis and deviations in hematologic and biochemical indices between survivors and non-survivors.

Ethical Clearance and Client Consent

This research was approved by the University of Ibadan Animal Care and Use Research Ethics Committee (UI–ACUREC/App/03/2017/007). Verbal consent was also obtained from the clients.

Conflict of Interest

The authors have no conflict of interest to declare.

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