RESEARCH ARTICLE

Haemato-Biochemical Studies on Chronic Gastritis in Dogs

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Abstract

This study was aimed to estimate the haemato-biochemical alterations in dogs suffering from chronic gastritis presented to Teaching Veterinary Clinical Complex of Faculty of Veterinary Sciences, Jammu for one year. A total of 30 clinically affected dogs presented with a history of chronic vomiting were included in the study and 6 healthy dogs were kept as a control group. Based on etiological factors, the diseased animals were divided into five groups: G1 (Foreign body), G2 (Renal failure), G3 (Hepatic origin), G4 (Diabetic origin), and G5 (Pyometra origin) with six animals in each group. Blood sampling was done on the day of presentation for all the groups to estimate haemato-biochemical parameters. Haemograms of dogs revealed low Hb, PCV and TEC in chronic gastritis due to renal, hepatic, diabetic and pyometra origin, and a significant increase in chronic gastritis due to gastric foreign body. A neutrophilic leukocytosis was observed in the foreign body, hepatic, diabetic, and pyometra groups, and neutropenia in the renal failure group. The biochemical parameters revealed a varied picture of reduced total protein and albumin and elevated BUN in the renal, diabetic, and pyometra groups. Creatinine and glucose levels revealed a significant increase in the renal and diabetic groups, respectively.

Key words: Chronic gastritis, Dog, Haematology, Serum biochemistry.

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Introduction

Vomiting persisting for longer than one or two weeks in dogs is clinically defined as chronic gastritis (Amorim *et al*., 2016). It is the chronic inflammation in gastric mucosa which is induced by a variety of factors, *viz.,* foreign body ingestion, dietary indiscretion, renal failure, hepato-biliary diseases, inflammatory bowel disease, occult parasitism, and *Helicobacter* spp. infection (Berghoff and Steiner, 2011; Amorim *et al*., 2016). It is a condition that results from inflamed stomach mucosal linings and is clinically characterized by episodes of abdominal pain, inappetence, sudden onset of vomiting, and weight loss (Patel *et al.,* 2018). It is common in dogs, with 35% of dogs evaluated for chronic vomiting and 26% to 48% of affected dogs remaining asymptomatic (Simpson, 2006). The diagnosis can be made based on clinical signs, laboratory findings, and results of imaging techniques such as radiography, ultrasonography, endoscopy, and histopathology. The disease has an unpredictable course and the diagnosis based on only clinical signs is challenging, hence cost-effective diagnostic tests are needed to determine the extra-gastrointestinal causes of gastritis. Therefore, the current study was performed to estimate some of the pertinent haematological and biochemical alterations that occur in dogs suffering from chronic gastritis, irrespective of the cause.

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Materials and Methods

The study was conducted at Small Animal OPD of Teaching Veterinary Clinical Complex, Faculty of Veterinary Sciences and Animal Husbandry, R.S Pura, SKUAST, Jammu, India from August 2020 to August 2021. Six clinically healthy adult dogs (age group 2 to 4 years) irrespective of breed and sex, presented for routine consultation (deworming and/or vaccination) or health check-ups at Clinics were randomly

selected as a control group (C) to determine the haemato-biochemical reference values. The clinically diseased dogs (n=30) presented with a history of chronic vomiting of more than one week duration were included, comprising 12 intact males, 8 neutered, 6 intact females, and 4 spayed, and were categorized into five groups (G1-Foreign body, G2-Renal failure, G3-Hepatic origin, G4-Diabetic origin, and G5-Pyometra induced) based on possible etiological factors after confirmation from detailed anamnesis, clinical findings, use of different diagnostic modalities, and histopathology.

Haemato-Biochemical Analysis

The blood samples were aseptically collected from the cephalic vein of dogs into EDTA vacutainers (2 mL) for the estimation of haematological parameters using a fully automatic haematology analyzer, and 2 mL in serum collecting vials for estimation of biochemical parameters. For serum separation, the blood samples were centrifuged immediately at 850 g for 15 min. The serum so obtained was transferred into Eppendorf tubes and preserved at -20°C until further analysis (Shyma and Vijayakumar, 2011) for various biochemical parameters by using commercially available Erba® diagnostic kits in Chem-7 semi-automated clinical chemistry analyzer.

The data obtained was subjected to one-way ANOVA using statistical package for the social sciences (SPSS 20 version). The results were expressed as mean and standard error (Mean ± SE). The statistical significance was assayed at 5% ($p<0.05$) and 1% ($p<0.01$) level.

Results and Discussion

The mean ± SE values and the statistical analysis of haemato-biochemical parameters are presented in Tables 1 and 2.

Table 1: Haematological findings in healthy and chronic gastritis affected dogs (Mean ± SE)

Parameters	Etiological groups of gastritis					
	Control (n=6)	G1 (n=6)	G2 (n=6)	G3 (n=6)	G4 (n=6)	G5 (n=6)
Hb (g/dL)	12.49 ± 0.18	14.23**± 0.19	9.02**± 0.16	9.17**± 0.13	10.07*± 0.39	10.20**± 0.05
TEC ($\times10^6$ µL)	5.94 ± 0.27	7.05**± 0.08	4.31**± 0.17	4.29**± 0.14	4.27* ± 0.22	4.96**± 0.07
PCV (%)	37.46 ± 0.53	42.69**± 0.58	27.05**± 0.49	27.50**± 0.39	30.21* ± 1.16	30.60**± 0.15
TLC ($\times10^3$/µL)	10.10 ± 0.66	21.09**± 0.96	16.04*± 0.33	15.52* ± 1.09	12.35*± 0.54	23.30**± 0.94
Neutrophils (%)	71.65 ± 0.49	82.74**± 0.70	62.79*± 0.60	78.21**± 0.46	75.98*± 0.44	79.66* ± 0.63
Lymphocytes (%)	22.11 ± 0.34	13.11* ± 0.20	31.00*± 0.63	17.61* ± 1.53	16.91*± 0.64	15.29* ± 0.47
Monocytes (%)	3.77 ± 0.31	1.89* ± 0.04	4.80 ± 0.43	2.52 ± 0.13	4.16 ± 0.33	1.92* ± 0.43
Eosinophil (%)	2.47 ± 0.22	2.26 ± 0.18	1.32* ± 0.14	1.89 ± 0.20	2.95 ± 0.04	1.21 ± 0.24
Basophil (%)	0.00 ± 0.00	0.00 ± 0.00	0.09 ± 0.06	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Platelets ($\times 10^5$ µL)	3.15 ± 0.30	3.60 ± 0.17	1.95** ± 0.10	2.11** ± 0.13	2.39** ± 0.34	2.12 ± 0.01

**Significant at $p<0.01$, *Significant at $p<0.05$ from control values.

Table 2: Serum biochemical findings in healthy and chronic gastritis affected dogs (Mean ± SE)

Parameters	Etiological groups of gastritis					
	Control (n=6)	G1 (n=6)	G2 (n=6)	G3 (n=6)	G4 (n=6)	G5 (n=6)
AST (IU/L)	32.04 ± 0.43	37.09 ± 1.84	41.94 ± 2.41	158.67**± 6.42	182.84* ± 2.67	55.82 ± 0.55
ALT (IU/L)	32.77 ± 0.56	26.96 ± 0.25	56.37 ± 1.33	143.89**± 4.90	184.90* ± 2.94	43.76 ± 1.75
ALP (IU/L)	72.57 ± 3.28	84.34 ± 1.41	112.45 ± 5.31	291.58**±18.16	296.84* ± 8.67	155.38 ± 0.39
Bilirubin (mg/dL)	0.67 ± 0.12	0.71 ± 0.08	0.68 ± 0.04	1.77** ± 0.25	0.96* ± 0.08	0.60 ± 0.02
Glucose (mg/dL)	90.67 ± 1.02	88.06 ± 0.93	85.00 ± 1.03	71.02**± 0.92	426.62**±42.21	86.26 ± 0.46
Total Protein (g/dL)	6.50 ± 0.11	7.08* ± 0.06	6.14 ± 0.37	6.31 ± 0.05	6.13 ± 0.08	7.81** ± 0.27
Albumin (g/dL)	3.79 ± 0.09	5.03* ± 0.04	2.15** ± 0.10	2.38** ± 0.11	2.27** ± 0.11	3.01** ± 0.05
Globulin (g/dL)	2.71 ± 0.11	2.05 ± 0.05	3.99* ± 0.37	3.93 ± 0.16	3.86* ± 0.04	4.80** ± 0.13
BUN (mg/dL)	18.52 ± 0.52	19.39 ± 0.57	140.5**± 2.40	20.02 ± 0.98	51.62* ± 0.85	72.29** ±3.34
Creatinine (mg/dL)	0.98 ± 0.03	1.02 ± 0.02	9.44** ± 0.86	1.44 ± 0.11	1.25 ± 0.07	2.12** ± 0.06
Calcium (mg/dL)	9.42 ± 0.11	9.48 ± 0.14	8.20* ± 0.17	9.71 ± 0.12	9.80 ± 0.80	9.45 ± 0.05
Phosphorus (mg/dL)	4.03 ± 0.08	4.58 ± 0.15	10.70** ± 0.87	4.62 ± 0.14	4.53 ± 0.41	3.99 ± 0.15

**Significant at $p<0.01$, *Significant at $p<0.05$ from control values.

G1 (Foreign body): A highly significant (p<0.01) increase was observed in the mean values of Hb, TEC, PCV, and TLC in dogs with foreign body induced gastritis as compared to the control group. The dehydration associated with vomiting leads to haemoconcentration further elevating haemoglobin, PCV, and TEC (Cornell and Koenig, 2016). Neutrophilic leucocytosis might be due to stress or inflammation associated with the foreign body in stomach. The biochemical profile revealed a significant (p<0.05) increase in total protein and albumin levels, which could be due to dehydration. Similar findings were documented by Cornell and Koenig (2016).

G2 (Renal failure): In comparison to the control group, a significant (p<0.01) decrease was recorded in the mean values of Hb, TEC, and PCV in dogs with renal failure induced gastritis. This might be related to uraemia, which shortens the survival period of RBC's leading to haemolysis, blood loss in the gastrointestinal tract and deficiency of erythropoietin synthesis (Tantary *et al.,* 2014; Sumit *et al.,* 2018). TLC in dogs with renal failure induced gastritis recorded a significant (p<0.05) increase when compared to control group. Leukocytosis may be due to the primary inflammatory diseases of kidneys and other body tissues. A significant (p<0.01) decrease was observed in the mean total platelet count in G2 group. The decreased platelet count might be due to inefficient thrombopoietic activity in chronic kidney disease or uraemic intoxication (Sumit *et al*., 2018). The mean value of ALP was on higher side, which might be due to secondary renal hyperparathyroidism. Similar observations were recorded by Sumit *et al.* (2018). The mean values of BUN and creatinine recorded a highly significant (p<0.01) increase in dogs with renal induced gastritis in comparison to the control group. The elevated levels of BUN and creatinine could be due to a considerable decrease in glomerular filtration rate (GFR), increased tubular urea absorption, and impaired renal capacity to excrete nitrogenous wastes. A highly significant (p<0.01) decrease in albumin levels might be due to renal or gastrointestinal loss of albumin, however, mean value of globulin recorded a significant (p<0.05) increase accounting for the normal total protein levels in our study. During the progress of an infection, marked changes occur in the serum proteins and the liver starts to synthesize acute phase proteins (APP's) mostly globulin (Kaneko *et al.,* 2008). The calcium levels revealed a significant (p<0.05) decrease in dogs with renal failure in comparison to control group which might be due to decreased absorption of calcium in the intestines owing to decreased calcitriol production from the damaged kidneys (Sumit *et al*., 2018). The mean value of phosphorus was significantly (p<0.01) elevated owing to decreasing renal function as kidneys are the principal route of phosphorus excretion in dogs (Puri *et al*., 2015).

***G3 (Hepatic origin)*:** The hepatic group revealed a significant (p<0.01) decrease in Hb, TEC, and PCV when compared with control group. This may be attributed to the increased red blood cell degeneration owing to the prolonged transit time of erythrocytes via spleen in response to lower portal blood flow or elevated red blood cell fragility in response to high bile acid concentration in the circulation (Rothuizen and Meyer, 2000). A significant (p<0.05) increase in the mean TLC was observed in this group in comparison to control group, which agreed with Tantary *et al.* (2014) and Lakshmi *et al.* (2018). The neutrophils were significantly (p<0.01) increased which could be due to the ongoing acute inflammatory conditions. This finding agreed with Lakshmi *et al.* (2018). A significant (p<0.01) reduction in platelet count was observed in gastritis of hepatic origin owing to the decreased thrombopoietin production by the liver, elevated platelet sequestration in the spleen, and increased breakdown of platelets (Lakshmi *et al.,* 2018). The mean values of AST, ALT, and ALP observed a highly significant (p<0.01) increase in this group as compared to control group, which could be due to inflammation, alteration of hepatic cell membrane permeability and necrosis leading to leakage of hepatocellular markers (Lakshmi *et al*., 2018). Alkaline phosphate is produced in response to bile retention and is considered as a cholestatic marker (Webster, 2010). The total bilirubin was significantly (p<0.01) elevated in comparison to control group which was in agreement with the report of Lakshmi *et al.* (2018). This might be due to the incapability of liver for bilirubin uptake, its conjugation, and excretion (Sevelius and Jonsson, 1995). A highly significant (p<0.01) decrease was recorded in glucose and albumin values in dogs with gastritis of hepatic origin in comparison to control group. Hypoglycemia might be due to inappetence/anorexia exacerbated by intestinal malabsorption and a marked reduction in liver function (Webster, 2010). The decreased albumin could be due to compromised hepatic function resulting in decreased synthesis and catabolism of plasma proteins. The mean value of total protein recorded a non-significant decline due to a marked anorexia, malabsorption, and protein-losing enteropathies, *viz.,* chronic gastritis, and gastroenteritis as also reported by Tantray *et al.* (2014).

G4 (Diabetic origin): The haemogram revealed a significant (p<0.05) decrease in the mean values of Hb, and PCV levels in dogs with gastritis of diabetic origin in comparison to the healthy control group. In our study, concurrent hepatic and renal involvement in diabetic dogs might be the reason for decreased Hb, and PCV which is in agreement with Hiblu *et al*. (2015). However, contrary to our study, Suresh (2009) observed increased PCV and Hb levels in diabetic dogs due to haemoconcentration along with persistent vomiting. A significant (p<0.05) elevation in the total leukocyte count (TLC) observed might be due to the ongoing inflammatory process, which concurred with Kasabalis *et al.* (2015). The mean total platelet count showed a significant (p<0.01) decrease in dogs with gastritis of diabetic origin, which could be due to inefficient thrombopoietic activity. A significant (p<0.05) increase was recorded in the mean values of AST, ALT, and ALP in dogs with gastritis of diabetic origin in comparison to control group. The

elevated liver enzyme activity may be secondary to hypoxia, hypovolemia, and concurrent diseases such as diffuse hepatic parenchymal changes leading to altered permeability of hepato-cellular membrane and inflammation (Jena *et al*., 2019). A highly significant ($p<0.01$) increase in mean value of blood glucose was observed which could be due to insulin deficiency and defective uptake of blood glucose by the peripheral tissues (Ghaffari *et al.*, 2008). The mean BUN value revealed a significant ($p<0.01$) increase with a non-significant increase in creatinine, which could be due to dehydration caused by vomiting, anorexia, unregulated glycosuria, and acidosis. The findings were in agreement with Jena *et al*. (2019). A significant ($p<0.01$) decline was recorded in albumin value due to mild liver cell damage in all the diabetic dogs. The mean value of globulin recorded a significant ($p<0.05$) increase in dogs with gastritis of diabetic origin, which might be due to urinary tract infections which increased globulin fractions.

G5 (Pyometra origin): A significant ($p<0.01$) decrease in mean levels of Hb, PCV, and TEC were recorded in dogs with pyometra induced gastritis in comparison to control group. This might be because of decreased erythropoiesis, loss of erythrocytes into the uterine lumen and toxic depression of bone marrow (Maharathi *et al.,* 2020). The mean value of TLC was significantly ($p<0.05$) decreased in this group in comparison to the control group. The decreased leukocyte count may be due to an inflammatory response and diffuse suppurative inflammation of uterus to combat the ongoing infection (Babu *et al*., 2018). A neutrophilic leukocytosis was found due to uterine infection having a chemotactic effect on neutrophils resulting in increased granulopoiesis. A significant ($p<0.01$) increase was recorded in the mean serum creatinine, BUN, total protein and globulin values and a significant ($p<0.01$) decrease in albumin in dogs with pyometra induced gastritis. The inability of kidneys to remove nitrogenous waste products under the influence of endotoxemia might be responsible for increased BUN level and increased serum creatinine may be due to hydration and pre-renal azotemia (Babu *et al*., 2018). The acute phase reaction is associated with increased synthesis of globulin in response to the bacterial infection. The low level of albumin in dogs with gastritis of pyometra origin might be attributed to chronic inflammation and a compensatory reduction in albumin as there might be renal loss of albumin (Maharathi *et al.,* 2020).

Conclusion

The present haemato-biochemical study revealed the increased level of Hb, TEC, and PCV in chronic gastritis due to gastric foreign body, whereas Hb, PCV and TEC were low in dogs with chronic gastritis due to renal, hepatic, diabetic and pyometra origin. A neutrophilic leukocytosis was observed in the foreign body, hepatic, diabetic, and pyometra groups and neutropenia in the renal failure group. The biochemical parameters revealed a varied picture with a decline in total protein and albumin and a significant elevation in BUN in dogs with chronic gastritis due to renal, pyometra and diabetic origin. A significant increase in creatinine and glucose values was observed in the renal and diabetic groups, respectively.

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