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Research Article

STUDY OF ALTERATION IN HAEMATO-BIOCHEMICAL INDICES IN CANINE GASTRITIS

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ABSTRACT

Gastritis is one of most common clinical complication in canines. The disease has multifactorial etiology and on most occasions, the exact etiology remains undetermined. The present study is an attempt to study the alteration in hematological and biochemical parameters in gastritis to delineate the most consistence parameters for future use in diagnosis of gastritis besides conventional methods. The study was conducted on 20 dogs (5 healthy and 15 with gastritis) of either sex to evaluate clinico-haemato-biochemical parameters in clinical cases of gastritis. The animals were divided into two groups, in first group five clinically healthy dogs were taken and the second group having fifteen dogs showing symptoms of gastritis. For both the groups, blood sampling was done on the day of presentation to measure clinico-haemato-biochemical parameters. There was significant rise in Haemoglobin ($P<0.01$), Packed cell volume (PCV) ($P<0.01$), Total Leukocyte count ($P<0.05$), Neutrophil ($P<0.01$), Monocyte ($P<0.05$), Eosinophils ($P<0.05$), serum Histamine ($P<0.01$) and Cortisol levels ($P<0.01$) in diseased group. The study suggests that plasma histamine and cortisol could be proved helpful in diagnosis of canine gastritis.

Keywords: Biochemical, Canines, Gastritis, Hematological

Gastritis is an outcome of inflamed gastric mucosal linings and clinically manifested as sudden onset of vomiting, loss of body weight, inappetance, and bouts of abdominal pain (Patel *et al.*, 2018). The development of gastritis is often attributed to development of immune responses against invading intraluminal antigens of dietary or bacterial origin, however, the exact underlying causes are rarely identified in majority of cases (Wiinberg *et al.*, 2005). Gastrin-17 and Histamine are essential hormones required for gastric mucosal cellular homeostasis. The elevated Gastrin-17 and Histamine promotegastric acid secretion and intensify gastric mucosal injury by reduction in blood flow, disturbed epithelial cell layer restoration, and reduced mucus secretions by releases of inflammatory mediators (Guarsio *et al.*, 2009 and Fourmy *et al.*, 2011). Besides, stress and exercise-inducedinduced elevated cortisol level may be having confounding effects on development of gastritis in the dogs (Monika *et al.*, 2018).The histological examination of tissues of gastric mucosais gold standard for the diagnosis of gastritis (Weigt *et al.*, 2020). Besides, the abdominal x-rays, ultrasonography and endoscopy are other useful tools for diagnosis. However, the ultrasonography and endoscopy requiring specialized instrumentation and cost intensive and histology being time intensive, there is need to identify the cost effective, fast and sensitive parameters for diagnosis. The present study was undertaken to evaluate diagnostic potential of haemato-biochemical parameters for clinical cases of gastritis.

MATERIALS AND METHODS

The present study was conducted on dogs presented to Referral Veterinary polyclinic, Indian Veterinary Research Institute, Izatnagar, Bareilly, Uttar Pradesh. Five healthy dogs were kept as control (Group 1). A total of fifteen dogs (Group-2), irrespective of age and sex presented with history of in appetence, vomiting, melena and abdominal pain were enrolled in the study. Group 1 dogs (n=5) were normal healthy dogs without any clinical signs of gastritis. The important clinical parameters of all the dogs were recorded in the form of questionnaire like inappetance/anorexia, vomiting, hematemesis, melena and abdominal pain. The blood sample was collected aseptically on the day of presentation (day 0) from saphenous/cephalic vein in EDTA vials and immediately transported to laboratory for hematology. The plasma samples were harvested on centrifugation of blood samples at 3000 rpm for 10 minutes and preserved at -200C for subsequent analysis. The hematological parameters such as haemoglobin (Hb), packed cell volume (PCV), total erythrocyte count (TEC), total leukocyte count (TLC), and differential leukocyte count were measured as per Jain, 1986. The enzymatic estimations for the study of biochemical parameters like total plasma proteins, Albumin, SGPT, SGOT, blood urea nitrogen (BUN) and creatinine were estimated by semi-automatic clinical chemistry analyzer with ready to use kits from Coral, Tulip Diagnostic Limited. Gastrin, Cortisol, Pepsinogen A and Pepsinogen C levels were estimated by ELISA method using Canine specific ELISA Kit from Genxbio Health Sciences Pvt. Ltd. Histamine level was estimated by Canine (HIS) ELISA Kit by Sun Red Biological Technology Co., Ltd. Thiobarbituric acid reactive substances (TBARS) were estimated by colorimetric assay procedure using Quanti Chrom TM TBARS Assay Kit® of Bioassay system and total antioxidant capacity (TAC) levels were estimated by colorimetric assay procedure

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usingtotal antioxidant capacity (TAC)Assay Kit® (C) from Bio Vision incorporation.

The data were statistically analyzed using SPSS software version 20. Significant differences among the groups were evaluated with one-way analysis of variance (ANOVA). All the statistical analyses were considered significant with $P<0.05$ and highly significant at $P<0.01$.

RESULTS AND DISCUSSION

Based on history and clinical examination as shown Table 1, all the 5 healthy control dogs were not having any symptom of gastritis, while all the 15 diseased dogs were showing different clinical signs of gastritis like anorexia/inappetence, vomiting with blood (n=3), with bile (n=0) and only gastric contents (n=13), melena (n=5), abdominal pain (n=15) on the day of presentation. Dog with gastritis were showing clinical signs viz. vomiting, hematemesis, melena, anorexia/inappetence, hypersalivation, licking of cold water, pain on palpation of abdomen and a specific praying posture due to gastric hyperacidity (Suchodolski *et al.*, 2005).

Haematological parameters (Mean±S.E.) of healthy control and dogs with gastritis on day of presentation are presented in Table 2. The Hb, PCV (%) and TLC count level was significantly ($P<0.05$) higher in group-2 than group-1. The Mean±S.E. of neutrophils (%), eosinophils (%) and monocyte (%) was significantly ($P<0.05$) higher in group-2 than group-1. Comparative analysis revealed no significant differences in the TEC, lymphocytes and basophils in group 1 and 2. Change in hematocrit value could be attributed to dehydration due to vomiting with loss of the body fluids lead to haemoconcentration which may occur in gastritis (Lidbury *et al.*, 2009). The decreased hematocrit, microcytic and hypochromic anaemia associated with severe gastric erosions and ulceration are being reported (Monika *et al.*, 2018). Anaemia with marked neutrophilia and a left shift occurred more frequently in dogs with chronic clinical conditions

Table 1

Comparison of clinical signs among both the groups

Groups	Clinical signs and symptoms					
	Anorexia	Vomiting			Melena	Abdominal pain
		Gastric contents with blood	Gastric contents with bile	Gastric contents		
Healthy control (n=5)	Absent	Absent	Absent	Absent	Absent	Absent
Diseased dogs (n=15)	15	03	00	13	05	15

Table 2

Haematological parameters of Healthy control and diseased dogs (Mean±S.E.)

Haematological Parameters	Healthy control (n = 5)	Diseased (n = 15)	P value
Hb (g/dl)	11.81±0.22	16.37±0.41	<0.05
PCV (%)	34.87±0.68	49.39±1.51	<0.01
TEC (106/ µl)	5.16±0.21	9.74±0.16	NS
TLC (103/µl)	6.8±0.83	13.33±0.74	<0.05
Differential leucocyte count			
Neutrophils (%)	70.2±0.8	75.8±2.15	<0.01
Lymphocytes (%)	29.2±0.58	21.33±2.41	NS
Monocytes (%)	0.6±0.4	1.47±0.34	<0.01
Eosinophils (%)	0±0	1.2±0.33	<0.05
Basophils (%)	0±0	0±0	NS

NS= Non- significant

(Fitzerald *et al.*, 2017). Contrarily, Stanton and Bright (1989) reported the normal leukogram in dogs with gastric diseases.

Serum biochemical parameters (Mean±S.E.) of healthy control and diseased dogs are summarized in Table 3. The plasma cortisol and histamine concentrations were significantly ($P<0.05$) higher in group-2 than group-1. The plasma concentration of SGPT, SGOT, BUN, creatinine, total Protein, albumin, Gastrin-17, Pepsinogen A and Pepsinogen C did not differ significantly in group-1 and group-2. No significant statistical difference was observed in SGOT, SGPT, Total protein, albumin, BUN and serum creatinine in diseased and healthy control dogs. However, in contrast to the present study, hyperproteinemia was recorded by Monika *et al.* (2018) which may be due to blood loss, liver disease, renal disease, malabsorption, or combination of the gastric diseases.

Significantly increased serum cortisol levels in the dogs with gastritis is suggestive of ongoing inflammatory responses. Stress induced increased endogenous cortisol production has been recorded in gastritis (Monika *et al.*, 2018). In the present study, significant increase in serum histamine levels was recorded. The significantly elevated histamine level was reported in dogs with gastritis (Ishiguro *et al.*, 2003). Histamine binds to receptors on the surface of the parietal cell produce c-AMP, leads to stimulating effects on gastric acid secretion from the apical H+–K+ATPase (Guariso *et al.*, 2009). H2 receptor-mediated signal which is required for gastric parietal cell homeostasis overcome cause impaired acid secretion (Kobayashi *et al.*, 2000).

The present study revealed that there were no significant differences in the serum gastrin, PGA and PGC

Table 3

Biochemical Parameters in Healthy Control and Dogs with gastritis (Mean±S.E.)

Biochemical Parameters	Healthy control (n=5)	Diseased (n=15)	P value
SGOT (IU/L)	23±1.7	29.4±1.47	NS
SGPT (IU/L)	33±3.03	35.87±1.93	NS
BUN (mg/dl)	21.4±2.46	19.27±1.44	NS
Creatinine (mg/dl)	0.9±0.14	0.93±0.06	NS
Total Protein (mg/dl)	6.78±0.21	5.69±0.21	NS
Albumin (mg/dl)	2.84±0.15	2.71±0.12	NS
Gastrin-17 (ng/L)	25.00±3.08	14.92±1.52	NS
Cortisol (µg/dl)	0.59±0.062	4.76±0.25	<0.05
Pepsinogen A (µg/L)	5.95±0.21	8.58±0.32	NS
Pepsinogen C (µg/L)	3.73±0.28	5.36±0.21	NS
Histamine (ng/ml)	0.73±0.05	5.90± 0.31	<0.01

NS= Non- significant

of healthy control and dogs with gastritis. However, in another study, the decreased serum gastrin-17 level has been reported which may be due to the excessive acid secretion leading to negative feedback mechanism that inhibits excessive gastrin secretion (Parante *et al.*, 2014; Monika *et al.*, 2018). However, it was also reported that the mean level of gastrin 17 was increased with the extension of gastritis because high number of gastric lesions leads to the greater stimulation of G cells (Hajsheykholeslami *et al.*, 2008). PGA is the predominant type of PG in dogs. The mean concentration of PGA and PGC was found non-significantly increased in dogs with gastritis of this study. The PGA and PGC was reported to be increased in gastritis (Suchodolski *et al.*, 2002). Low serum PGA and a low PGA/PGC ratio have been recognized as useful diagnostic biomarkers for the corpus atrophic gastritis (AG), screening to risk of gastric neoplasm and other extra gastric diseases so that high serum PG concentrations indicates not only the gastric dysfunction but also the other abnormalities (Monika *et al.*, 2018).

CONCLUSION

Plasma histamine and cortisol levels could be used as sensitive biomarkers in diagnosis of canine gastritis along with conventional methods. Further, a detailed study on plasma PGA and PGC on large number of samples is warranted for ascertaining the usefulness in the diagnosis of canine gastritis.

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