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Haemato-biochemical alterations and effect of herbal-probiotics combination regimens in acute gastritis in young dogs

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Abstract
In the present study, the haematological-biochemical investigation was done on 24 dogs suffering from acute gastritis. The clinical signs in the affected dogs were observed vomiting, anorexia, abdominal pain and dehydration. Haematology revealed non-significant difference in all the parameters. In biochemical alterations, significant changes in potassium were observed and non-significant changes were seen in alanine aminotransferase, total protein albumin, globulin, chloride and sodium and potassium. The affected dogs were randomly divided into four groups. the group 1 served as the healthy control, group 2 was treated with *Zingiber officinale* aqueous extract @ 100 mg/kg + *Lactobacillus acidophilus* + *Sacharomyces boulardii* @ 2 g PO, OD for 5 days, group 3 was treated with Fennel seeds aqueous extract @ one teaspoonful + *Lactobacillus acidophilus*+ *Sacharomyces boulardii* @ 4 g PO, OD for 5 days and group 4 was treated with *Elettaria cardamomum* aqueous extract @ one teaspoon+ Lactobacillus acidophilus + Sacharomyces boulardii @ 6 g PO, BID for 5 days. The present investigation showed that herbal therapeutic regimen in group 2 was most effective for the treatment of acute gastritis as compared to group 3 and group 4.

Keywords: Acute gastritis, haematological-biochemical analyses, herbal-probiotics, dog

Introduction
Gastritis representing Inflammation of gastric mucosa, gastritis is commonly manifested in vomiting. In dogs gastric disorders usually include mucosal inflammation, ulceration, obstruction, or neoplasia and the categorization is based on the grade of intensity of gastritis depending on the nature of cellular infiltration and histo-architectural anomalies (Day *et al*., 2008) [3]. Gastritis is broadly classified into acute and chronic forms. Clinically, acute gastritis is characterized by the sudden episode of vomiting, whereas chronic gastritis involves intermittent vomiting lasting over a period of 1-2 weeks or more. Gastritis often leads to in-appetance, loss of body weight, and repeated bouts of abdominal pain (Patel *et al*., 2018) [7].
In most dog patients, the causes include accidental exposure to hazardous toxins, e.g. plant pesticides and insecticides, heavy metals, and bleaching agents, dietary indiscretion, injudicious drug therapy, *viz*. non-steroid anti-inflammatory drugs (NSAIDs), corticosteroids, and antibiotics, (Ettinger and Feldman 2010) [5]. Ingestion of foreign bodies may obstruct the pyloric region of stomach and result in acute clinical symptoms. However, stagnation of the foreign object may precipitate mucosal trauma with acute gastritis, systemic disease, uremia, liver dysfunction or hypo-adrenocorticism (Amorim *et al*., 2016) [1].

Materials and Methods
A total of 328 dogs of different breeds, presented at Teaching Veterinary Clinical Service Complex, Jabalpur suffering from gastritis were evaluated. Detailed clinical manifestations and clinical parameters were recorded in 24 dogs selected for therapeutic trial. About 2 ml blood sample was collected aseptically from the cephalic or saphenous vein on day 0 pre-treatment, and on day 3 and day 6 post treatment from each dog and transferred into clean, dry EDTA glass vials for routine haematology. One ml aliquot of the blood sample was transferred into clot activator vials and serum was harvested carefully after centrifugation (3000 rpm/ minute, 5 minutes), frozen and stored (-20°C) for biochemical analysis. Haemoglobin (Hb), packed cell volume (PCV), total erythrocyte count (TEC), total leucocyte count (TLC) and differential leucocyte count (DLC) were determined using standard methods. The biochemical parameters such as serum alanine transferase (ALT) (U/l), serum total protein (g/dl),

serum albumin (g/dl), serum globulin (g/dl), serum chloride (mEq/l), serum sodium and potassium (mEq/l) were estimated in serum on day 0 (pre-treatment) and on days 3 and 6 (post treatment) with the help of semi auto analyzer by using readymade kit manufactured by Erba, Manheim, Transasia biomedical (India) PVT LTD. The affected dogs were randomly divided into four groups. the group 1 served as the healthy control, group 2 was treated with *Zingiber officinale* aqueous extract @ 100 mg/kg + *Lactobacillus acidophilus* + *Sacharomyces boulardii* @ 2 g PO, OD for 5 days, group 3 was treated with Fennel seeds aqueous extract @ one teaspoonful + *Lactobacillus acidophilus*+ *Sacharomyces boulardii* @ 4 g PO, OD for 5 days and group 4 was treated with *Elettaria cardamomum* aqueous extract @ one teaspoon+ Lactobacillus acidophilus + Sacharomyces boulardii @ 6 g PO, BID for 5 days.

Results and Discussion

Examination of the haematological profile (Hb concentration, TEC and PCV %) did not reveal onset of anaemic state in the dog patient. However, Papazoglou *et al.* (2003) [6]. Reported elevation in the PCV %, presumably because of generalized tissue dehydration. Thus, it is concluded that the intensity of acute gastritis in the dog patient (present study) is of only mild to moderate intensity. Further prevention of tissue dehydration in treatment points to efficacy of all the therapeutic combination regimens. Leucocytosis with concurrent increase in neutrophil % is dependable marker of pathogenic bacterial infection in the GIT. No significant increase in the values of TLC and neutrophil % clearly indicate the absence of microbial infection in the dog patients (present study). Further, absence of lymphopenia and eosinopenia suggest pre-emption of stress in the patients through effective treatment. It is well-known that eosinophilia is a reliable bio-marker of parasitic infection and allergic conditions. However, it is noteworthy that there was no incidence of eosinophilia pre-treatment in this study. This is a positive sign of proper home management of pets by the well-informed clients.

Table 1: Haemoglobin (g/dl) in different treatment groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	13.11±0.52	13.36±0.45	13.85±0.42
2	T_2	12.85±0.44	11.96±0.46	12.01±0.45
3	T_3	13.16±0.52	12.30±0.44	12.56±0.52
4	T_4	12.00±0.76	11.68±0.70	11.78±0.70

Table 2: Mean packed cell volume (%) in different treatment groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	40.96±0.78	40.5±0.75	40.71±0.85
2	T_2	39.78±2.67	37.6±3.01	38.16±2.69
3	T_3	41.40±0.71	40.23±0.87	40.06±1.20
4	T_4	39.80±1.01	38.23±0.87	38.53±0.72

Table 3: Mean total erythrocyte count ($\times 10^6$/µl) in different treatment groups

S. No.	Group	Day 0	Day 3	Day 6
1	T1	5.88±0.51	5.87±0.45	5.99±0.62
2	T2	5.21±0.38	4.56±0.40	4.86±0.38
3	T3	5.18±0.28	4.58±0.30	4.66±0.32
4	T4	4.05±0.38	3.45±0.30	3.90±0.30

Table 4: Mean total leukocyte count ($\times 10^3$/µl) in different treatment groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	11.17±0.84	11.38±0.98	11.33±0.95
2	T_2	12.21±0.80	11.70±0.80	11.15±0.76
3	T_3	12.18±2.30	11.63±2.32	11.08±2.30
4	T_4	11.51±1.13	10.93±0.97	10.71±0.85

Table 5: Mean neutrophil (%) in different treatment groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	80.83±2.42	80.50±2.72	80.33±2.64
2	T_2	81.33±1.56	80.50±1.58	80.16±1.83
3	T_3	81.00±3.28	80.33±3.07	80.00±3.08
4	T_4	81.50±2.04	80.33±2.18	80.83±1.11

Table 6: Mean lymphocyte (%) in different treatment groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	12.83±0.79	12.66±1.05	12.50±0.99
2	T_2	14.00±1.06	13.33±1.11	12.50±0.51
3	T_3	12.66±0.95	12.16±1.35	12.00±1.39
4	T_4	13.16±0.79	12.83±1.66	12.16±1.16

Table 7: Mean monocyte (%) in different treatment groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	5.16±0.60	5.50±0.56	5.00±0.73
2	T_2	5.66±0.66	5.33±0.66	5.16±0.54
3	T_3	6.50±0.56	5.33±0.61	5.50±0.22
4	T_4	5.50±0.56	5.16±0.30	5.00±0.57

Table 8: Mean eosinophil (%) in different treatment groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	3.33±0.55	3.50±0.92	3.66±0.98
2	T_2	3.66±0.55	3.33±0.42	3.16±0.47
3	T_3	4.00±0.51	3.83±0.47	3.33±0.66
4	T_4	3.83±0.47	3.50±0.56	3.66±0.42

Table 9: Mean alanine aminotransferase (u/l) in different treatment groups at different intervals

S. No.	Group	DAY 0	DAY 3	DAY 6
1	T_1	38.39±2.84	38.83±2.82	38.86±2.97
2	T_2	43.35±2.76	42.45±2.73	40.81±2.77
3	T_3	48.70±4.70	47.35±4.59	46.25±4.56
4	T_4	45.80±3.59	44.91±3.70	43.25±3.72

Table 10: Mean total protein (g/dl) in different treatment groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	6.38±0.40	6.06±0.38	6.11±0.44
2	T_2	7.09±0.41	6.70±0.30	6.31±0.18
3	T_3	6.93±0.29	6.65±0.30	6.58±0.30
4	T_4	7.21±0.44	6.80±0.42	6.51±0.46

Table 11: Mean albumin (g/dl) in different groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	3.65±0.26	3.56±0.30	3.36±0.31
2	T_2	4.04±0.21	3.98±0.18	3.48±0.19
3	T_3	3.86±0.21	3.68± 0.21	3.63±0.20
4	T_4	4.18±0.17	4.05±0.19	3.61±0.20

Table 12: Mean globulin (g/dl) in different groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	2.73±0.22	2.50±0.21	2.75±0.33
2	T_2	3.05±0.23	2.85±0.24	2.80±0.20
3	T_3	3.06±0.12	3.02±0.22	2.95±0.14
4	T_4	3.03±0.32	2.90±0.30	2.85±0.17

Table 13: Mean chloride (mEq/l) in different groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	112.88±1.33	113.02±1.23	112.61±1.19
2	T_2	105.90±1.53	107.06±1.71	108.56±1.94
3	T_3	104.98±0.78	105.85±1.30	106.48±1.30
4	T_4	105.27±0.81	106.03±0.77	107.57±1.03

Table 14: Mean sodium (mEq/l) in different groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	141.11±1.09	141.05±1.37	141.60±1.27
2	T_2	137.89±5.25	139.47±5.05	141.05±4.8
3	T_3	138.08±2.27	139.02±2.25	139.99±2.30
4	T_4	139.23±0.55	140.37±0.58	140.89±0.88

Table 15: Mean potassium (mEq/l) in different groups at different intervals

S. No.	Group	Day 0	Day 3	Day 6
1	T_1	4.31±0.23	4.62±0.20	4.55±0.19
2	T_2	5.04^a±0.20	4.50^b±0.12	4.43^b±0.07
3	T_3	4.93±0.31	4.65±0.26	4.63±0.20
4	T_4	5.03±0.24	4.66±0.19	4.56±0.16

Changes in the values of blood-biochemical parameters carefully reflect alterations in homeostasis for example, increased alanine aminotransferase (ALT) titre signify damage to functional hepatocytes. Sagar (2016) [10] reported significantly increased values in dogs suffering from gastritis and increasing trend, though statistically not significant, was also observed in the present study.

Apparent hyper-proteinaemia in vomiting dogs reflects generalized tissue dehydration thus, Sagar (2016) [10]. Observed hyper-albuminaemia with non-significant increase in the total protein concentration in blood circulation. It is reasonable to presume that cell water loss was minimized in response to the effective herbal-probiotics-fluid replacement combination therapies.

Maintenance of *in vivo* electrolyte balance is of primordial significance. It is well-known that where as sodium Na^+ is mainly extracellular, potassium ($K^{+)}$ is mostly intracellular. On the contrary, the serum potassium (K^+) titer in the gastritis dogs in group T2 was significantly higher (P<0.05) *vs.* the healthy control group T1 (Table 15) pointing to hyperkalemia. This observation is in complete agreement with the earlier report (Sagar, 2016) [10]. Similar observation was reported by Gennari and Weise (2008) [8]. .Hyperkalemia observed in the present investigation may be attributed to increased retention of K^+ ions by kidney parenchyma and tubular reabsorption. Hypochloremia is generally associated with hyponatremia in the present study, hypochloremia was a salient feature of vomiting dogs pre-treatment (day 0). Hypochloremia may be due to loss of chloride ions and acid rich gastric secretions through vomiting as suggested by Hoskins *et al.* (1998) [9]. However, the anions imbalance was effectively restored following the combination regimens restored towards normalcy.

Conclusion

An overview of the haemato-biochemical profile of the gastritis affected dogs pre-treatment (day 0) and post treatment (day 3 and 6) clearly suggests the best therapeutic response is elicited with ginger *Zingiber officinale*, Hindi 'adrak' (group T2), closely followed by cardamom *Elettaria cardamomum* Hindi 'elaichi' (group T4) Fennel seeds *Foeniculum vulgare* (saunf) was also effective though to a less effective as compared to T2 and T4.

In gastritis episodes in dogs metabolic acidosis is the outcome of anaerobic micro-environment resulting from hypoxia and subsequent metabolic alkalosis to the accelerated loss of H^+ into the gastric lumen (Broom and Walsh, 2003) [2]. Further hypovolaemic shock and deranged acid based balance in metabolic acidosis/ alkalosis may lead to life-threatening situations (Elwood, 2010) [4].

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