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Research Article

Investigations of pancreas functions in dogs with gastritis

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ABSTRACT

The aim of the study was to determine whether pancreatitis develops in dogs with chronic gastritis. It was also aimed to investigate energy metabolism in dogs with chronic gastritis. Gastritis may occur primarily or secondarily due to diseases of surrounding organs and systems. Complications such as pancreatitis, hepatitis and nephritis may develop due to gastritis. In this study, gastroscopy was performed on dogs with suspected chronic gastritis and 20 dogs with gastric lesions were included in the study. In addition, 10 clinically healthy dogs with no gastroscopic lesions were used as the control group. Addionally, dogs with chronic gastritis were divided into two groups: hemorrhagic and non-hemorrhagic. Blood samples were collected from all dogs and serum samples were prepared and stored at -80oC until use. Canine specific amylase (AML), pancreatic lipase (cPL), insulin (INS), asprosin (ASP), glucose (GLU) and total cholesterol (total-C) values were measured in these serum samples. In the study, serum levels of cPL ($p<0.05$), INS ($p<0.01$), ASP ($p<0.01$), GLU ($p<0.05$) and total-C ($p<0.01$) were significantly higher in dogs with chronic gastritis than the values of dogs in the control group. There was no significant difference in AML levels of dogs with gastritis and control group. According to the cut-off values, increases in cPL, AML, INS, ASP, GLU and total-C values were determined in 12 (%60), 8 (%40), 15 (%75), 15 (%75), 11 (%55) and 13 (%65) dogs, respectively. While cPL values of dogs in the hemorrhagic gastritis group were determined to be statistically higher than those in the non-hemorrhagic group, increases in cPL values were also determined in more animals in this group. Additionally, a moderate positive correlation ($p<0.05$) was determined between cPL and ASP; a high positive correlation ($p<0.01$) was determined between AML and total-C, and between GLU and ASP. In conclusion, the results of the study showed that pancreatitis may develop in some dogs with chronic gastritis. In addition, increases in asprosin, insulin and total-C levels in dogs with chronic gastritis indicate that energy and lipid metabolisms are also affected.

INTRODUCTION

Gastritis is an inflammation of the gastric mucosa and may develop primarily due to causes directly affecting the gastric mucosa or may be secondary to diseases of other organs or systems. Systemic diseases, ulcerogenic or irritating drugs, gastric foreign bodies, fungal infections and infections of organs such as liver and kidney, immune deficiencies (atrophic gastritis), congenital disorders (hypertrophic gastritis), food allergy, inflammatory bowel disease, parasites and hypereosinophilic syndrome play roles in the etiology of gastritis (Blois,2025; Patel et al, 2020; Simpson, 2013; Steiner, 2025).

The pancreas is a digestive organ that produces hormones such as insulin and secretes enzymes. It becomes acutely or chronically inflamed due to a variety of causes including gastritis, enteritis, gastroenteritis, cancers of the pancreas or surrounding organs, and gastrointestinal foreign bodies. Pancreatitis in dogs can be caused by feeding fatty pork, beef and other fatty human foods. It can also be caused by dogs eating food from garbage cans. Some drugs, viral and bacterial infections are also among the causes of pancreatitis (Bansal et al., 2019; Steiner, 2025). When pancreatitis occurs, the pancreas releases enzymes and other substances into the abdominal area, causing local inflammation. These substances damage the pancreas and surrounding organs, causing life-threatening complications. Pancreatic enzymes also concentrate within its own tissue, destroying it and causing pancreatitis to become more severe. Sometimes pancreatitis can also cause diabetes by disrupting the pancreas' ability to regulate blood sugar by secreting insulin (Bansal et al., 2019; Steiner, 2025).

The clinical signs of gastritis in dogs are very similar to those of pancreatitis and it is extremely difficult to differentiate these two diseases clinically. Both the stomach and the pancreas can be secondarily infected from surrounding organ infections. Studies in humans have shown that gastritis and pancreatitis can develop simultaneously in the same patient (Bansal et al., 2019; Hartung, 2018; Tositti, 2001, Steiner, 2025). Similarly, a case report study in dogs revealed that gastritis and pancreatitis developed simultaneously in dogs (Chan, 2006; Stainer, 2025). The development of pancreatitis in dogs with chronic gastritis and the status of energy metabolism in dogs with chronic gastritis have not been sufficiently investigated

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to date. Thus, the aim of the study was to determine whether pancreatitis develops in dogs with chronic gastritis and whether pancreatic functions are affected. Additionally, the status of energy metabolism in these dogs was also investigated.

MATERIALS and METHODS

Dogs

Twenty dogs with suspected gastritis and clinical signs such as vomiting, abdominal distension, abdominal pain and irregular appetite for more than 15 days were used in the study. Gastroscopy was performed on the dogs and dogs with gastric lesions were included in the study. In addition, 10 clinically healthy dogs with no gastric lesions on gastroscopy were used as the control group. Dogs of different sexes and breeds over 1 year of age were included in the study.

Clinical analysis

In the study, information such as disease duration, clinical findings, and age were obtained from the animal owner and recorded. These dogs were then subjected to routine clinical examinations, and heart rate, respiratory rate, rectal temperature, and symptoms such as vomiting, diarrhea, abdominal distension, and abdominal pain were recorded.

Gastroscopy

Gastroscopy was performed on all the dogs in both groups (Spużak et al., 2020; Zoran, 2001). For gastroscopy procedures, dogs were pre anesthetized with atropine at a dose of 1 mg/kg body weight (bw) intravenously (IV) (Xylazinbio 2%, Interhas) and 0.05 mg/kgbw intramuscularly (IM) (Vetaş Atropine 0.2%, Vetaş). Ten minutes after this procedure, ketamine 2-4 mg/kgbw (Ketasol 10%, Interhas) was administered IV (Brearley et al., 2006). Gastroscopy (EPX 3500HD endoscopy device, Japan) was performed in 20 minutes and images of the identified lesions were recorded. According to the lesions determined, 20 dogs with gastritis were also divided into two groups as hemorrhagic (n=9) and non-hemorrhagic gastritis (n=11).

Blood samples

Blood samples were collected from all dogs via the cephalic vein into tubes without anticoagulant and used to prepare serum samples. These serum samples were stored at −80°C until further use.

Biochemical analysis

Serum insulin [Canine (INS) ELISA kit, SunRed, catolog no: 201-15-0201, Shanghai, CHINA] and asprosine (ASP, Canine Asprosine ELISA kit, MyBiosource; MBS2612398, San Diego, USA) levels were measured using canine specific double-antibody sandwich ELISA kits. ELISA test kits were performed according to the procedure recommended by the manufacturer. Pancreas specific lipase (Canine pancreatic lipase; cPL, rapid quantitative test, Healvet Veterinary immunofluorescence Quantitative Analyzer, CHINA) was analyzed in dog sera by quantitative hormone analyzer. In addition, amylase (AML), glucose (GLU) and total cholesterol (Total-C) were also analyzed with a biochemistry device (MScan II rotor; Melet Schloesing M-Scan II, FRANCE).

In the study, in order to calculate asprosine and insulin values in the sera of dogs, two fold dilutions of the standard insulin (80mU/L-6.25mU/L) and asprosine (10ng/mL-0.156ng/mL) supplied with the ELISA kits were used. Regression analysis was performed with the OD values obtained from these dilutions and a standard curve graph was created and the curve fit line was drawn. Formulas were created for the y-axis in this graph and these formulas were used to calculate insulin ($y=66.459x-32967$, $R^2=0.998$) and asprosin ($y=66.459x-32967$, $R^2=0.998$) values in serum samples.

Statistical analysis

The normality of the distribution of the data was examined with the Kolmogorov-Smirnov test. Statistical differences between parameters obtained from healthy dogs and dogs with gastritis were analyzed by two-way Student's t test. One-way ANOVA (posthoc Duncan) or Kruskal Wallis H tests for parameters with high variation were used to determine statistical differences between data from control, hemorrhagic gastritis and non-hemorrhagic gastritis dogs. In addition, Perason's Correlation Coefficint (r) test was used to determine the correlation between the data. ROC analysis was performed for each parameter and cut-off values were determined. To determine individual increases, the parameter of each dog was compared with the cut-off value and the parameter above the cut-off value was accepted as an increase. Statistical significance level was accepted as $p<0.05$ in the evaluations. SPSS 27.0 for Windows® package program was used for the tests.

RESULTS

Clinical findings

In the anamnesis and clinical examinations of the dogs in which gastritis was detected by gastroscopy in the study, it was found that there were signs of loss of appetite, lying on cold places, abdominal tension, abdominal pain, weakness, weight loss and occasional vomiting for more than 15 days.

Gastroscopy findings

No lesions were detected in healthy dogs on gastroscopy (Figure 1a,b). Gastroscopy revealed hemorrhagic gastritis of various types and sizes in 9 of the 20 dogs with suspected gastritis (Figure 2a,b) and non-hemorrhagic gastritis (diffuse inflammatory 5, ulcerative 3, atrophic 2, hypertrophic 1, n=11) in 11 dogs (Figure 3a,b).

Biochemical parameters

In the study, serum cPL ($p<0.01$), INS ($p<0.01$), ASP ($p<0.01$), total-C ($p<0.01$) and GLU ($p<0.05$) oncentrations were significantly higher in dogs with gastritis compared to healthy dogs (Table 1). However, no statistical difference was found between AML levels in healthy dogs and dogs with gastritis (Table 1).

When the parameters of dogs with hemorrhagic and non-hemorrhagic gastritis were compared, INS ($p<0.01$) and

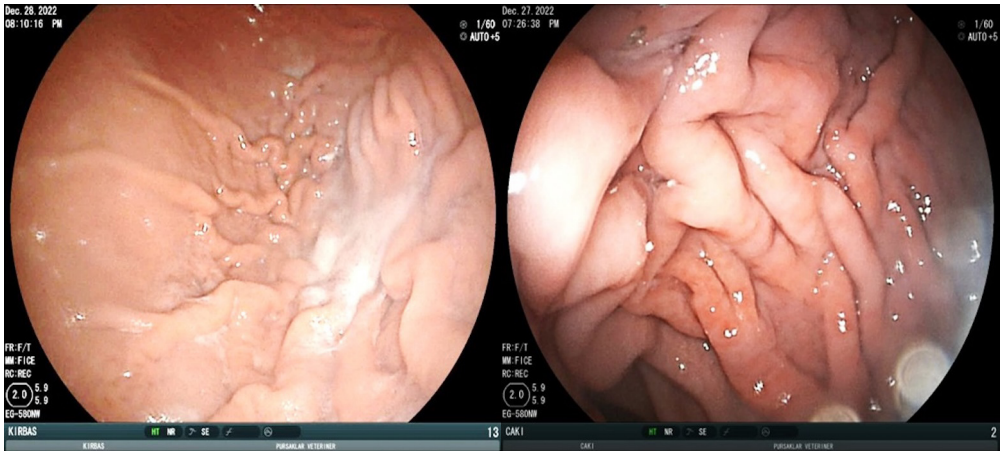

Figure 1. Gastroscopy images of healthy dogs

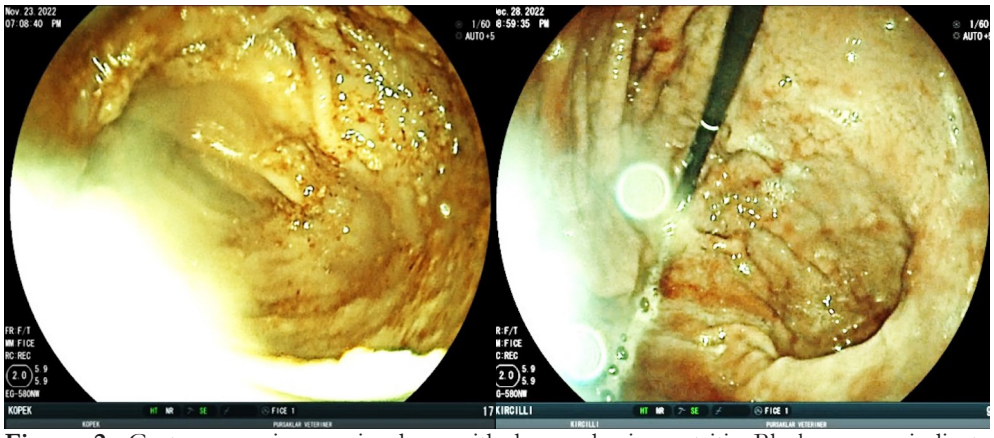

Figure 2. Gastroscopy images in dogs with hemorrhagic gastritis Black arrows indicate hemorrhagic areas

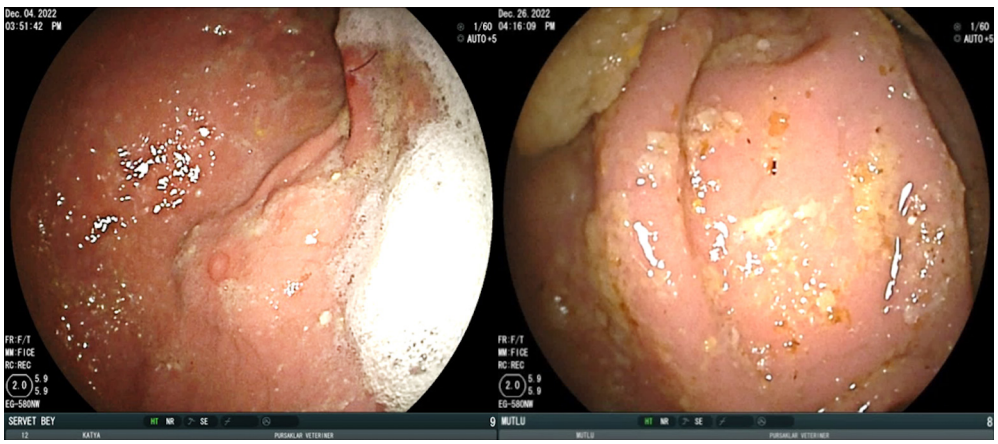

Figure 3. Gastroscopy images in dogs with ulcerative non-hemorrhagic gastritis. Black arrows indicate ulcerative areas

ASP ($p<0.01$) values were higher in both groups compared to the control group, but there was no significant difference between them (Table 2). The cPL values of dogs in the hemorrhagic gastritis group were higher than both control and non-hemorrhagic gastritis groups ($p<0.01$). The total-C values of the dogs with non-hemorrhagic gastritis were higher than the control group ($p<0.05$), but not significantly different from the dogs with hemorrhagic gastritis (Table 2).

According to the cut-off values, serum cPL concentrations was increased in 12 (60%) and AML in 8 (40%) dogs with gastritis (Table 3). Furthermore, GLU, INS, ASP and total-C values were determined to be increased in 11(55%), 15(75%), 15(75%), 15(75%) and 13(65%) dogs with gastritis, respectively (Table 3). The cPL levels of 8(%40) and 3(%15) dogs with gastritis ranged from 200 to 400 µg/L (suspected pancreatitis) and higher than 400 µg/L (pancreatitis), respectively. The cPL

levels of the 9(%45) dogs with gastritis in the study were determined to be below 200 µg/L, which is considered normal. Additionally, more dogs in the hemorrhagic gastritis group (%100) had an increase in cPL compared to the non-hemorrhagic group (%27.27). The numbers and rates of dogs with increased cPL, AML, INS, ASP, GLU and total-C values in dogs with gastritis, hemorrhagic and non-hemorrhagic gastritis are shown in Table 3.

Table 1. Serum pancreatic lipase (cPL), serum amylase, INS, ASP, GLU and Total-C levels in healthy dogs and dogs with gastritis.

Parameters	Control (n=10)	Gastritis (n=20)	Min-Max	P value
cPL (µg/L)	178.96±77.39	240.05±105.83	46.69-501.95	0.007
AML (U/L)	715.66±364.78	911.6±430.91	242-2147	0.134
INS (mU/L)	17.27±7.4	32.8±13.04	7.3-54.07	0.002
Asprosin (ng/mL)	1.78±0.31	2.73±0.86	1.33-4.72	0.003
GLU (mg/dL)	93.75±29.06	111.45±19.71	52-147	0.03
Total-C (mg/dL)	241.5±104.97	377.2±122.32	131-522	0.005

Table 2. Serum cPL, AML, INS, ASP and GLU levels in healthy dogs and dogs with hemorrhagic and nonhemorrhagic chronic gastritis.

Parameters	Control (n=10)	Hemorrhagic gastritis (n=9)	Nonhemorrhagic gastritis (n=11)	Medyan (Min-Max)
cPL (µg/L)	178.96±77.39 [a]	316.83±101.2 [b]	177.24±58.37 [b]	200.69 (46.69-501.95)
AML (U/L)	715.66±364.78[a]	899.11±336.82[a]	921.81±511.65 [a]	865 (242-2147)
INS (mU/L)	17.27±7.4 [a]	30.83±12.03 [b]	34.42±14.17 [b]	26.48 (7.3-54.07)
Asprosin (ng/mL)	1.78±0.31 [a]	2.73±0.86 [b]	2.6±0.65 [b]	2.24 (1.33-4.72)
GLU (mg/dL)	93.75±29.06 [a]	111.45±19.71 [a]	111.81±23.21 [a]	108.5 (52-147)
Total-C (mg/dL)	241.5±104.97 [a]	377.2±122.32 [ab]	402.27±108.64 [b]	357.5 (131-522)

cPL: canine pancreatic lipase, AML: Amylase, INS: Insulin, ASP: Asprosin, GLU: Glucose, Total-C: Total cholesterol. The degree of statistical significance between groups is determined by letters, and the presence of different letters in the same line indicates statistical significance between groups ($p<0.05$).

Table 3. Number and percentage values (%) of dogs with chronic gastritis (Hemorrhagic+Nonhemorrhagic), hemorrhagic and nonhemorrhagic chronic gastritis according to cut-off values.

	Cut-off value	Gastritis (Hemorrhagic+ Nonhemorrhagic) (n=20)	Hemorrhagic Gastritis(n=9)	Nonhemorrhagic Gastritis (n=11)
cPL (µg/L)	188.28	12(%60)	9(%100)	3(%27.27)
AML (U/L)	865	8(%40)	4(%44.44)	4(%36.36)
INS (mU/L)	21.55	15(%75)	6(%66.66)	9(%81.81)
ASP (ng/mL)	2.09	15(%75)	7(%77.77)	8(%72.72)
GLU (mg/dL)	107.5	11(%65)	5(%55.55)	6(%54.54)
Total-C (mg/dL)	292.5	13(%65)	5(%55.55)	8(%72.72)

cPL: canine pankreatic lipaz, AML: Amilaz, İNS: İnsülin, ASP: Asprosin, GLU: Glukoz, Total-C: Total kolesterol

Correlation analysis revealed a moderate positive correlation ($p<0.05$) between cPL and ASP, a high positive correlation ($p<0.01$) between AML and total-C ($p<0.01$), and between GLU and ASP ($p<0.01$, Table 4).

Table 4. Number and percentage values (%) of dogs with chronic gastritis (Hemorrhagic+Nonhemorrhagic), hemorrhagic and nonhemorrhagic chronic gastritis according to cut-off values.

	cPL (µg/L)	AML (U/L)	INS (mU/L)	ASP (ng/mL)	GLU (mg/dL)	Total-C (mg/dL)
cPL (µg/L)	1	-0.135	0.257	0.419*	-0.087	-0.110
AML (U/L)		1	0.293	0.101	0.104	0.503**
GLU (mg/dL)			1	0.633**	0.168	0.284
ASP (ng/mL)				1	0.069	0.075
INS (mU/L)					1	0.042
Total-C (mg/dL)						1

cPL: canine pancreatic lipase, AML: Amylase, INS: Insulin, ASP: Asprosin, GLU: Glucose, Total-C: Total cholesterol
The degree of significance of the correlation between the parameters is indicated by symbols. *: p<0.05, **:p<0.01.

DISCUSSION

Gastritis is defined as inflammation of the stomach mucosa and is frequently seen in dogs. It is caused by foreign, non-food substances, inappropriate or allergenic foods, toxic substances, and a variety of medications such as non-steroidal anti-inflammatory drugs (NSAIDs) and steroids. It can also occur due to bacterial, viral and parasitic infections as well as hepatitis and pancreatitis. (Blois, 2025; Patel et al, 2020; Simpson, 2013). For this reason, the pancreas is often affected by gastrointestinal tract infections or inflammations (Alagappan et al., 2022; Bansel et al., 2019; Hartung, 2018; Steiner, 2025). In studies, chronic gastritis has also been found to be quite common in patients with chronic pancreatitis (Bansal et al., 2019; Chan, 2006; Hartung, 2018; Tositti, 2001; Steiner, 2025). The symptoms seen in pancreatitis are very similar to the clinical findings in dogs with chronic gastritis, it is extremely difficult to distinguish them clinically. Therefore, the diagnosis of pancreatitis is very difficult and all information obtained from X-ray, ultrasound, hematology, biochemistry and biopsy should be utilized. Apart from these, more specific tests such as pancreatic lipase immunoreactivity (PLI) test and canine specific pancreatic lipase (cPL) are used to diagnose pancreatitis in dogs (Lim et al., 2022; Steiner et al., 2008; Steiner, 2025; Trivedi et al., 2011).

In the present study, gastroscopy was performed on the dogs and hemorrhagic and non-hemorrhagic lesions of varying numbers and diameters were observed. Gastroscopy findings revealed that the lesions were quite diverse, including hemorrhagic, diffuse inflammatory, erosive, ulcerative, atrophic, hypertrophic, in accordance with previous studies in dogs with chronic gastritis symptoms (Amorim et al., 2016; Blois, 2025; Kim et al., 2020). It was thought that this diversity may vary depending on the etiology, severity and process of gastritis.

Pancreatic lipase is produced by pancreatic acinar cells, and its levels in the blood increase significantly during pancreatitis (Lim et al., 2022; Steiner, 2025; Trivedi et al., 2011). In the studies and commercial test kits (idexx), the reference values for cPL are; cPL≤200 µg/L is considered normal, cPL≥400 µg/L is considered pancreatitis and 200-399 µg/L is considered suspicious for pancreatitis (Lim et al., 2022; Steiner, 2025; Trivedi et al., 2011).

Amylase is an enzyme produced mainly by the pancreas, but also to a lesser extent by the salivary gland, small intestines and liver. Blood amylase levels may increase with factors such as anorexia, ulcerative gastritis, pancreatitis, stress, chronic renal failure, hepatitis, trauma, salivary gland inflammation and certain drug use (metranidazole, furasemide). It is suggested that pancreatitis should be suspected when blood levels increase 3 times the normal values (Cridge et al., 2021; Tositti, 2001). For these reasons, serum amylase levels are considered moderately effective in the diagnosis of pancreatitis (Steiner et al., 2008; Wallig and Sullivan, 2013; Willard and Twedt, 2009).

In the present study, high cPL values were determined in dogs with both chronic gastritis (p<0,05) and hemorrhagic gastritis (p<0,01) compared to healthy dogs (p<0.05). While, no statistical difference was found between the AML levels of the all groups. However, according to the cut-off values, it was determined that cPL increased in 12 (60%) and AMY increased in 8 (40%) dogs with chronic gastritis. cPL was found to be increased in a greater number of dogs with hemorrhagic gastritis than in dogs with non-hemorrhagic gastritis. In dogs with hemorrhagic gastritis, the proportion of dogs with suspected pancreatitis (cPL: 200-400 µg/L) and those with pancreatitis (cPL>400 µg/L) was determined as 66.66% and 27.27%, respectively. As a result of the present study, the increases in blood AML and cLP levels may not be present in all dogs with chronic gastritis and this may be related to the gastritis process and severity. Studies in dogs and humans have shown that pancreatitis often develops with gastrointestinal tract infections or inflammations (Alagappan et al., 2022; Bansal et al., 2019; Hartung, 2018; Tositti, 2001; Steiner, 2024). Therefore, it can be said that pancreatitis may develop or coexist in some dogs with chronic gastritis and pancreatic functions may be affected accordingly (Steiner et al., 2008; Wallig and Sullivan, 2013; Willard and Twedt, 2009).

Blood glucose levels are maintained by a balance between hormone that lower glucose (insulin) and hormones that increase it (glucagon, cortisol, epinephrine, norepinephrine and growth hormone) (Hantzidiamantis et al., 2025; Idowu and Heading, 2018). In the fasting state, the normal glucose level is maintained by endogenous sources (glycogen), while in the fed state, glucose is taken directly from exogenous sources (Hantzidiamantis et al., 2025; Idowu and Heading, 2018). Or-

gans such as the liver, pancreas, adrenal gland, thyroid gland and anterior pituitary gland play important roles in blood glucose regulation. The liver acts as a glucose storage organ. In case of excess carbohydrate intake and elevated blood glucose levels, unusable glucose is stored as glycogen in the liver and skeletal muscles, and when glucose is needed, glucose is produced from these stores and released into the blood. The pancreas provides intracellular and extracellular regulation of blood sugar levels. In case of an increase in blood sugar, insulin is secreted from the islets of Langerhans β cells of the pancreas and the blood sugar level is lowered. When blood glucose levels decrease, glucagon is secreted by the α cells of the islets of pancreatic langerhans and glycogen stores are stimulated to release glucose into the blood (Hantzidiamantis et al., 2025; Idowu and Heading, 2018). Excessive glucose consumption in the body, malnutrition, endocrine or hepatic disorders, chronic renal failure, malabsorption, maldigestion, severe infection or sepsis, hypoadrenocorticism and insulinoma can lead to low blood glucose levels (Breitschwerdt et al., 2014; Idowu and Heading, 2018). Moderately elevated blood glucose levels (hyperglycemia) occur as a result of stress, infections (teeth, kidneys, bladder, sepsis), inflammatory conditions (pancreatitis) and hormonal imbalances (hyperadrenocorticism), while persistent high hyperglycemia cases occur as a result of diabetes mellitus (Hagley et al., 2020; Hantzidiamantis et al., 2025; Idowu and Heading, 2018).

Chronic gastritis and pancreatitis often occurs in association with gastrointestinal infections or inflammations (Alagappan et al., 2022; Bansal et al., 2019; Hartung, 2018; Tositti, 2001). Hyperglycemia, hyperinsulinemia, and hypercholesterolemia associated with inflammation, infection, and sepsis have been reported in dogs with gastritis and pancreatitis (Alagappan et al., 2022; Bansal et al., 2019; Hagley, 2020; Moosavian and Fazli, 2022; Tositti, 2001). In the present study, high levels of INS ($p<0.01$), ASP ($p<0.01$), GLU ($p<0.0$) and total-C ($p<0.01$) were obtained in dogs with chronic gastritis. Glucose, INS, ASP and total C values were determined to be high in 13 (65%), 11 (55%), 15 (75%) and 15 (7%) dogs with chronic gastritis, respectively. Additionally, moderate positive correlations were determined between cPL and ASP ($p<0.05$) and high positive correlations ($p<0.01$) were determined between AML and total-C, and GLU and ASP. Hyperglycemia, hyperinsulinemia and hypercholesterolemia determined in the present study may have developed due to severe stress, inflammation, infection and pancreatitis, which are frequently encountered in gastrointestinal diseases (Blois, 2025; Moosavian and Fazli, 2022; Simpson, 2013).

Anorexia, hunger and vomiting frequently occur in dogs with chronic gastritis and pancreatitis (Alagappan et al., 2022; Bansal et al., 2019; Blois, 2025; Hartung, 2018; Tositti, 2001). It is well-known that, asprosin is produced from adipose tissue to increase blood glucose levels during starvation and anorexia. Asprosin stimulates both glucose production from the liver and increased appetite via the pituitary gland (Duerrschmid et al., 2017; Keser and Ünüsan, 2021; Romere et al., 2016). Plasma asprosine levels are significantly linked to glucose metabolism, lipid profile, insulin resistance (IR) and β-cell function (Farrag et al., 2023; Lee et al., 2019; Sarıkaya and Gökce, 2024). Plasma asprosin levels have been found to be significantly higher in the fasting state, which has been explained by increased asprosin production from the liver to stimulate glucose release and appetite. (Lee et al., 2019; Romere et al., 2016). The high asprosin values detected in our study are probably due to increased asprosin production by the liver to stimulate glucose production and appetite in anorexia and fasting conditions seen in gastritis (Alagappan et al., 2022; Bansal et al., 2019; Lee et al., 2019; Romere et al., 2016).

In conclusion, it was determined that the levels of pancreas-specific lipase, total cholesterol, insulin, glucose and asprosin were high in dogs with chronic gastritis. Pancreatitis developed in some dogs with chronic gastritis and pancreatic functions were affected accordingly. It is conceivable that lipid and energy metabolism are affected in dogs with chronic gastritis.

CONCLUSION

The results of the present study showed that pancreatitis may be observed in some dogs with chronic gastritis. Changes in pancreas-specific lipase, asprosin, insulin, glucose, and total cholesterol levels suggest that both pancreatic function and energy metabolism are affected in some dogs with chronic gastritis. The development of pancreatitis and changes in energy metabolism are thought to be related to the severity and duration of gastritis. Therefore, pancreatitis should be taken into consideration in the diagnosis and treatment of chronic gastritis in dogs.

DECLARATIONS

Ethics Approval

This study was approved by the Animal Ethics Committee (AEC), Burdur Mehmet Akif University, Türkiye (No:930/2022).

Conflict of Interest

Authors do not have any conflict of interest fort his study.

Consent for Publication

Consent on publication was confirmed with approval from the Republic of Türkiye Ministry of Agriculture and Forestry, Directorate of Burdur Provincial (No: E-69877819-325.04.02-5917085).

Author contribution

Idea, concept and design: HİG, YEA, Data collection and analysis: HİG, YEA, Drafting of the manuscript: HİG, YEA, Critical review: HİG, YEA

Data Availability

Not applicable.

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