Himachal Journal of Agricultural Research
Volume 52, Issue 1, June 2026; pp. 208–213; DOI: 10.18311/HJAR/2026/80345

INFORMATICS JOURNALS
Veterinary Sciences

Clinical, Hemato-biochemical and Ultrasonographic Alterations in Dogs with Pancreatitis and their Therapeutic Management

Jagarapu Vamsi Krishna[1]*, Ankur Sharma[1], Ajay Katoch[1], Pardeep Sharma[1] and Deepti Sharma[2]

[1]Department of Veterinary Medicine, DGCN College of Veterinary and Animal Sciences, Chaudhary Sarwan Kumar Himachal Pradesh Krishi Vishvavidyalaya, Palampur, Himachal Pradesh, India; vamsikrishnajagarapu@gmail.com
[2]Department of Veterinary Surgery and Radiology, DGCN College of Veterinary and Animal Sciences, Chaudhary Sarwan Kumar Himachal Pradesh Krishi Vishvavidyalaya, Palampur, Himachal Pradesh, India

Abstract

The present study evaluated clinical, hematobiochemical and ultrasonographic alterations in dogs suffering from pancreatitis and their therapeutic management. Out of 3344 dogs presented to the Veterinary Clinical Complex during the study period, 24 dogs were diagnosed with pancreatic disorders, of which six dogs were diagnosed with pancreatitis. Common clinical signs observed were lethargy, persistent vomiting, anorexia, prayer's position and abdominal pain. Haematological evaluation revealed leukocytosis with neutrophilia and monocytosis, while biochemical analysis showed significant elevations in BUN, creatinine, ALP, amylase, lipase, LDH and C-reactive protein. Electrolyte imbalances, including hyponatremia and hypokalemia, were also recorded. Diagnosis was based on a combination of clinical signs, hematobiochemical alterations and ultrasonographic findings, including pancreatic enlargement and altered echogenicity, which collectively supported pancreatic inflammation and injury. However, SNAP cPL testing could not be performed due to the unavailability of the diagnostic facility. Therapeutic management resulted in clinical recovery in affected dogs.

Keywords: Amylase, Canine Pancreatitis, Inflammatory Response, Lipase, Ultrasonography

Pancreatic disorders in dogs are of increasing clinical importance due to their impact on digestive and metabolic functions[1]. The pancreas is a dual-function gland composed of endocrine and exocrine components. The endocrine portion regulates glucose homeostasis through hormones such as insulin and glucagon, whereas the exocrine portion produces digestive enzymes, including amylase and lipase, necessary for nutrient digestion[2].

Pancreatitis is one of the exocrine pancreatic disorders in dogs. It is characterised by inflammation of pancreatic tissue associated with premature activation of digestive enzymes within the gland, resulting in autodigestion of pancreatic tissue and systemic inflammatory response[1]. Various predisposing factors, including dietary indiscretion, obesity, hyperlipidemia, endocrine disorders such as diabetes mellitus and hyperadrenocorticism, certain drugs, trauma, and infectious agents, including bacterial, viral and parasitic pathogens, have been reported to contribute to the development of pancreatitis[2]. Clinically, affected dogs commonly present with anorexia, vomiting,

*Author for correspondence

Manuscript received: 30.03.2026; Accepted: 16.05.2026

abdominal pain, lethargy and dehydration, and the severity of the disease may vary from mild to severe systemic involvement[3].

Diagnosis of pancreatitis is based on clinical presentation supported by laboratory investigations such as serum amylase, lipase, C-reactive protein and lactate dehydrogenase estimation, along with ultrasonographic examination to identify structural pancreatic changes[4]. Early diagnosis and prompt supportive therapy, including fluid management, analgesia and dietary modification, are essential for improving clinical outcomes. Considering the increasing clinical importance of pancreatitis in dogs, the present study was conducted at the Veterinary Clinical Complex, DGCN College of Veterinary and Animal Sciences, to evaluate clinical, hematobiochemical, and ultrasonographic alterations in affected dogs, and to assess their response to therapeutic interventions.

MATERIALS AND METHODS

The present study was conducted at the Department of Veterinary Medicine in Veterinary Clinical Complex, DGCN College of Veterinary and Animal Sciences, CSKHPKV, Palampur (Himachal Pradesh) from May 2024 to December 2025. Dogs presented with clinical signs suggestive of pancreatitis, including anorexia, vomiting, abdominal pain upon palpation, prayer posture, lethargy and weakness, were included in the study. Diagnosis was based on detailed clinical examination supported by laboratory investigations and ultrasonography. Dogs diagnosed with pancreatitis based on clinical, laboratory and ultrasonographic findings were included in the study group. Apparently healthy dogs presented for routine examination were included as controls for comparative evaluation of laboratory parameters. A detailed history, including age, breed, sex, dietary pattern and clinical signs, was recorded for each case. Physical examination included recording of rectal temperature, mucous membrane evaluation, hydration status assessment and abdominal palpation.

Blood samples were collected aseptically through venipuncture of the cephalic or saphenous vein. Haematological parameters, including total leukocyte count, differential leukocyte count, erythrocytic indices (haemoglobin, packed cell volume, mean corpuscular volume, mean corpuscular haemoglobin, and mean corpuscular haemoglobin concentration), and platelet count, were analysed using an automated veterinary haematology analyser (Shenzhen Mindray Animal Medical Technology Co. Ltd.). Haematological parameters were measured by cytometry using reagents manufactured by Shenzen Mindray Animal Medical Technology, China. Serum biochemical parameters such as blood urea nitrogen, creatinine, total protein, albumin, bilirubin, glucose, AST, ALT and ALP were estimated using an automated chemistry analyser (Agappe Diagnostic Ltd.). The biochemical parameters were measured by spectrophotometry using reagents supplied by Agappe Diagnostics Ltd., 'Agappe Hills', Distt. Ernakulam, Kerala- 683 562. Serum amylase and lipase were measured using the IDEXX Catalyst One veterinary chemistry analyser. Lactate dehydrogenase was estimated by the photometric method, and C-reactive protein was determined using the ELISA technique. Serum electrolytes (Na^+, K^+ and Cl^-) were analysed using the ion-selective electrode method.

Abdominal ultrasonography was performed using a 5–7.5 MHz microconvex probe to evaluate pancreatic size, echogenicity and associated structural changes. Dogs diagnosed with pancreatitis were managed with medical therapy. Fluid therapy using Ringer's lactate or normal saline was administered intravenously at 40–60 ml/kg/day, depending on the hydration status, until dehydration was corrected. Antibiotic therapy included ceftriaxone–tazobactam (Inj. Intacef-Tazo) at 20–25 mg/kg IV every 12 h and metronidazole (Inj./Tab. Metrogyl) at 10–15 mg/kg IV/PO every 12 h for 5–7 days. Analgesia was provided using butorphanol (Inj. Butrum) at 0.05–0.1 mg/kg IV/IM/SC every 6–8 h for 3–5 days. Antiemetic therapy consisted of ondansetron (Inj. Emeset) at 0.1–0.2 mg/kg IV/IM every 8–12 h for 3–5 days. Pantoprazole (Inj. Pantosec) was administered at 1 mg/kg IV every 12 h for 5 days. Dietary management included low-fat, easily digestible diets offered in small, frequent meals once vomiting subsided. Therapeutic response

was assessed based on improvement in clinical signs such as appetite, vomiting and hydration status during hospitalisation.

RESULTS AND DISCUSSION

During the study period, a total of 3344 canine cases were presented to the Veterinary Clinical Complex, among which 24 dogs were diagnosed with pancreatic disorders based on clinical and diagnostic findings, of which six cases were diagnosed as pancreatitis. An equal occurrence of pancreatitis was observed among dogs belonging to the 4–6 years, >6–9 years and >9 years age groups, indicating that the condition may affect dogs across a wide age range, although middle-aged and older dogs are generally considered more predisposed to pancreatitis[1,2]. A higher proportion of affected dogs were males (83.33%) compared to females (16.66%). This finding is in partial agreement with previous reports suggesting a possible male predisposition in canine pancreatitis[5], although sex-related susceptibility remains inconsistent across studies. The most common clinical sign observed was lethargy (6/6), followed by persistent vomiting (5/6),

Table 1. Alterations in haematological parameters in diseased dogs (Mean ± SE)

S. No.	Parameters	Healthy (n=10)	Diseased Dogs (n=6)
1	TLC (× 10^3/µL)	11.31 ± 0.52	28.16 ± 8.29*
2	Neutrophils (×10^3/µL)	8.72 ± 2.53	23.18 ±7.28*
3	Lymphocytes (×10^3/µL)	1.91 ± 0.45	1.41 ± 0.58
4	Monocytes (×10^3/µL)	0.15 ± 0.13	3.05 ± 1.14**
5	Eosinophils (×10^3/µL)	0.14 ± 0.11	0.37 ± 0.13
6	Neutrophils (%)	77.09 ± 2.13	81.28 ± 4.93
7	Lymphocytes (%)	16.92 ± 0.05	6.23 ± 2.71
8	Monocytes (%)	1.32 ± 1.7	11.63 ± 4.66*
9	Eosinophils (%)	1.24 ± 0.9	1.53 ± 0.59
10	TEC (× 1012/L)	7.66 ± 1.7	5.05 ± 0.91
11	Hb (g/dL)	13.9 ± 1.33	12.18 ± 2.07
12	PCV (%)	42.01 ± 2.22	31.28 ± 5.53
13	MCV (fL)	59.22 ± 0.89	62.01 ± 1.26
14	MCH (pg)	22.25 ± 0.41	25.95 ± 1.47**
15	MCHC (g/dL)	36.66 ± 0.77	39.45 ± 1.15
16	RDW (%)	13.66 ± 0.06	16.25 ± 0.85**
17	PLT (× 109/L)	237 ± 2.33	277.66 ± 73.46

*p<0.05, **p<0.01, ***p<0.001

Table 2. Alterations in biochemical parameters in diseased dogs (Mean ± SE)

S. No.	Parameters	Healthy (n=10)	Diseased Dogs(n=6)
1	Glucose (mg/dL)	106.78 ± 2.33	106.47 ± 11.38 (61.7-143)
2	BUN (mg/dL)	24.55 ± 1.59	90.97 ± 35.02*
3	Creatinine (mg/dL)	0.71 ± 0.33	4.025 ± 1.84*
4	Total protein (g/dL)	6.89 ± 0.06	5.58 ± 0.57**
5	Albumin (g/dL)	2.49 ± 0.08	2.09 ± 0.34
6	Albumin – globulin ratio	1.3 ± 0.21	0.63 ± 0.12*
7	Total bilirubin (mg/dL)	0.14 ± 0.03	2.72 ± 2.46
8	AST (U/L)	27.48 ± 1.9	186.06 ± 120.07
9	ALT (U/L)	34.56 ± 1.18	172.74 ± 92.52
10	ALP (U/L)	36.72 ± 0.55	705.1 ± 367.8*

*p<0.05, **p<0.01, ***p<0.001

Table 3. Changes in electrolytes in diseased dogs (Mean ± SE)

S. No.	Parameters	Healthy (n=10)	Diseased Dogs (n=6)
1	Na^+ (mMol/L)	151.71 ± 1.66	141.55 ± 3.06**
2	K+ (mMol/L)	4.50 ± 0.15	3.60 ± 0.25**
3	Cl^- (mMol/L)	109.02 ± 1.49	104.5 ± 2.56

*p<0.05, **p<0.01, ***p<0.001

anorexia (4/6), prayer's position (4/6) and abdominal pain (3/6). These findings are consistent with previous reports describing lethargy, vomiting and abdominal pain as predominant clinical manifestations of canine pancreatitis[6].

Clinical evaluation of dogs with pancreatitis revealed mild alterations in vital parameters, characterized by a non-significant increase in heart rate (85.66 ± 4.49 bpm) and a significant increase in respiratory rate (37 ± 2.88 breaths/minute) compared to healthy controls (HR in healthy dogs: 76.8 ± 2.25 bpm; RR: 28.5 ± 2.2 breaths/min), which may be attributed to abdominal pain, dehydration and systemic inflammatory response. Rectal temperature showed mild variation but remained within physiological range. Similar tachycardia and tachypnea have been reported in dogs with acute pancreatic inflammation due to pain and inflammatory mediators[1,3].

Haematological evaluation revealed significant leukocytic alterations indicative of an inflammatory response in dogs with pancreatitis. A significant increase in total leukocyte count was observed, accompanied by marked neutrophilia and monocytosis, along with a significant rise in monocyte percentage compared to healthy controls. Among erythrocytic indices, Mean Corpuscular Haemoglobin (MCH) and Red Cell Distribution Width (RDW) were also found to be significantly elevated in affected dogs. Biochemical analysis demonstrated significant alterations in several parameters, including elevated Blood Urea Nitrogen

Table 4. Changes in amylase and lipase in diseased dogs (Mean ± SE)

S. No.	Parameters	Healthy (n=10)	Diseased Dogs (n=6)
1	Amylase (U/L)	808.66 ± 201.07	2256 ± 175.91***
2	Lipase (U/L)	753.66 ± 174.35	3522.33 ± 735.98**

*p<0.05, **p<0.01, ***p<0.001

Table 5. Alterations in LDH & CRP in diseased dogs (Mean ± SE)

S. No	Parameter	Healthy (n=10)	Diseased Dogs
1	LDH (U/L)	121.82 ± 22.16	237.5 ± 31.9* (n=4)
2	C-reactive Protein (mg/dL)	3.04 ± 0.90	8.5 ± 0.36** (n=5)

*p<0.05, **p<0.01, ***p<0.001

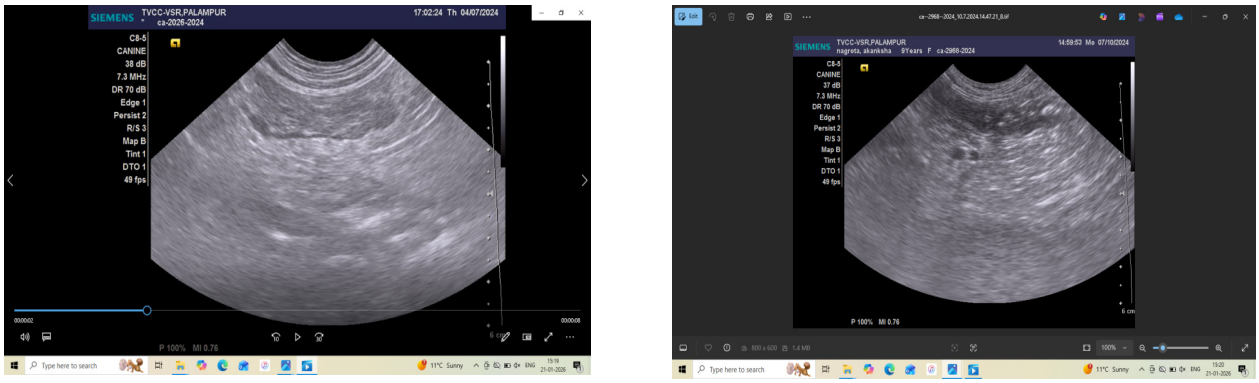


Figure 1. Ultrasonographic image showing hypoechoic Pancreas along with an increase in size.

(BUN), creatinine concentrations, suggestive of azotemia, which may be attributed to dehydration, reduced renal perfusion, and possible renal involvement associated with systemic inflammatory responses in canine pancreatitis and alkaline phosphatase (ALP) levels, while total protein levels were significantly reduced in diseased animals. In addition, the Albumin–Globulin (A/G) ratio was significantly decreased in affected dogs compared to healthy controls, indicating alterations in serum protein fractions associated with inflammatory processes. Urine analysis was only performed in two cases; therefore, a detailed correlation between azotemia and urinary findings could not be established in the present study. Furthermore, marked elevations in pancreatic enzymes were recorded in affected dogs. While healthy dogs showed normal serum amylase and lipase levels with respect to diseased dogs, dogs with pancreatic involvement demonstrated substantially higher enzyme activities, suggesting acinar cell injury and leakage of digestive enzymes into the circulation. Levels of lactate dehydrogenase (LDH) were also elevated, which may be attributed to cellular necrosis and systemic inflammatory response, as LDH is released from damaged pancreatic and surrounding tissues. Similar increases in pancreatic enzymes and LDH have been reported in canine pancreatitis[1,2,7]. Increased inflammatory markers, such as C-reactive protein, in the present study further supported systemic inflammatory activation. Electrolyte imbalances observed in affected dogs were most likely secondary

to persistent vomiting and dehydration, which are common sequelae of acute pancreatitis. However, confirmation of pancreatitis ideally requires specific diagnostic tests such as SNAP cPL, which was not performed in the present study.

Ultrasonographic evaluation revealed pancreatic enlargement and altered echogenicity in affected dogs (Plate 1). These findings are characteristic of inflammatory pancreatic changes and have been previously reported in clinical studies[4]. Following therapy, including fluid management, analgesics, antibiotics, and dietary modification, clinical improvement was observed in surviving dogs within 5–7 days of treatment, indicating a favourable response to early intervention.

Following the above medical management, including fluid therapy, analgesics, antibiotics and dietary modification, clinical recovery was reported in all six dogs post 15 days of treatment. All the dogs were alive till 6 months post-treatment.

REFERENCES

1. Watson P. Pancreatitis in dogs and cats: definitions and pathophysiology. J Small Anim Pract. 2015; 56(1):3-12. https://doi.org/10.1111/jsap.12293
2. Xenoulis PG, Steiner JM. Canine and feline pancreatic lipase immunoreactivity. Vet Clin Pathol. 2012; 41(3):312-24. https://doi.org/10.1111/j.1939-165X.2012.00458.x
3. Mansfield C. Acute pancreatitis in dogs: advances in understanding, diagnostics, and treatment. Top Companion Anim Med. 2012; 27(3):123-32. https://doi.org/10.1053/j.tcam.2012.04.003
4. Steiner JM. Diagnosis of pancreatitis. Vet Clin North Am Small Anim Pract. 2003; 33(5):1181-95. https://doi.org/10.1016/S0195-5616(03)00061-5
5. Hess RS, Kass PH, Shofer FS, Van Winkle TJ, Washabau RJ. Evaluation of risk factors for fatal acute pancreatitis in dogs. J Am Vet Med Assoc. 1999; 214(1):46-51. https://doi.org/10.2460/javma.1999.214.01.46
6. Pápa K, Máthé Á, Abonyi-Tóth Z, Sterczer Á, Psáder R, Hetyey C, *et al.* Occurrence, clinical features and outcome of canine pancreatitis (80 cases). Acta Vet Hung. 2011; 59(1):37-52. https://doi.org/10.1556/avet.59.2011.1.4
7. Hess RS, Kass PH, Ward CR. Breed distribution of dogs with diabetes mellitus admitted to a tertiary care facility. J Am Vet Med Assoc. 2000; 216(9):1414-7. https://doi.org/10.2460/javma.2000.216.1414

AUTHORS CONTRIBUTION

JVK: Conducted clinical investigations, collected and analyzed data, interpreted results, and prepared the original manuscript. AS: Conceived and supervised the study, provided academic guidance, interpreted data, and critically reviewed the manuscript. AK: Assisted in clinical examinations, case management, and interpretation of clinical findings. PS: Assisted in clinical investigations, case management, and manuscript review. DS: Performed and interpreted ultrasonographic examinations and contributed to manuscript review and editing.