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Manikantaswamy BM

Consultant Veterinarian, My
Pets Choice Veterinary Clinic,
Bengaluru, Karnataka, India

Ranjita Siddappa Rajur

M.V.Sc. Scholar, Department of
Veterinary Medicine, Veterinary
College, Bengaluru, Karnataka,
India

Anil Kumar MC

Professor, Department of
Veterinary Clinical Complex,
Veterinary College, Bengaluru,
Karnataka, India

Acute pancreatitis in a beagle: A case report

Manikantaswamy BM, Ranjita Siddappa Rajur and Anil Kumar MC

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Abstract

A five-year-old male Beagle dog was presented with a history of recurrent vomiting, anorexia, lethargy, abdominal pain, and progressive weakness. Clinical examination revealed dehydration, tachycardia, pyrexia, abdominal tenderness, and delayed capillary refill time. Hematological examination showed leukocytosis with neutrophilia and hemoconcentration, while serum biochemical analysis revealed elevated serum amylase, lipase, ALT, and ALP activities.

The case was presumptively diagnosed as severe acute pancreatitis based on compatible clinical signs, hematobiochemical alterations, elevated serum amylase and lipase activities, and therapeutic response. The animal was managed with aggressive intravenous fluid therapy, analgesics, antiemetics, proton pump inhibitors, antibiotics, and intensive supportive care. Gradual clinical improvement was observed following therapy with restoration of appetite and normalization of vital parameters.

The present case emphasizes the importance of early diagnosis, intensive monitoring, and prompt supportive therapy in the successful management of severe pancreatitis in dogs. Timely therapeutic intervention plays a vital role in reducing complications and improving clinical prognosis in affected canine patients.

Keywords: Acute pancreatitis, Beagle, canine, pancreatic inflammation, supportive therapy

Introduction

Pancreatitis is one of the most common disorders affecting the exocrine pancreas in dogs and is associated with considerable morbidity and mortality depending on the severity of inflammation (Mansfield, 2012) ^[5]. Acute pancreatitis is characterized by reversible inflammatory injury to the pancreas, whereas chronic pancreatitis involves irreversible histopathological changes including fibrosis and acinar atrophy (Watson, 2015) ^[12].

The pathogenesis of acute pancreatitis involves premature intracellular activation of digestive enzymes within pancreatic acinar cells leading to autodigestion, pancreatic inflammation, vascular injury, and tissue necrosis (Mansfield, 2012) ^[5]. In severe cases, inflammatory mediators including cytokines, complement proteins, kinins, and reactive oxygen species contribute to systemic inflammatory responses and multiorgan dysfunction (Mansfield, 2012) ^[5].

Pancreatic disorders in dogs are often associated with significant systemic inflammatory response and may rapidly progress to life-threatening complications if not recognized and managed promptly (Watson *et al.*, 2004; Abhijith *et al.*, 2025) ^[11, 1].

Clinical presentation of canine pancreatitis varies widely from mild gastrointestinal disturbances to severe life-threatening systemic disease (Cridge *et al.*, 2021) ^[3]. Common clinical signs include vomiting, anorexia, lethargy, dehydration, abdominal pain, diarrhea, pyrexia, and weakness (Mansfield, 2012) ^[5]. Severe forms of pancreatitis may result in systemic complications such as disseminated intravascular coagulation, acute kidney injury, cardiac arrhythmias, pulmonary edema, and shock (Mansfield and Beths, 2015) ^[6].

Several risk factors have been implicated in canine pancreatitis including dietary indiscretion, high-fat diets, obesity, endocrinopathies, hyperlipidemia, drug administration, and genetic predisposition (Cridge *et al.*, 2021; Abhijith *et al.*, 2025) ^[3, 1]. Certain breeds including Miniature Schnauzers, Yorkshire Terriers, Cocker Spaniels, and Beagles are reported to possess increased susceptibility to pancreatic disorders (Mansfield, 2012) ^[5].

Corresponding Author:

Manikantaswamy BM

Consultant Veterinarian, My
Pets Choice Veterinary Clinic,
Bengaluru, Karnataka, India

Diagnosis of acute pancreatitis remains challenging because no single diagnostic test can definitively confirm the disease in all cases (Cridge *et al.*, 2021) [3]. Hematological and biochemical abnormalities commonly include leukocytosis, hemoconcentration, elevated hepatic enzymes, azotemia, electrolyte imbalance, and increased pancreatic lipase concentrations (Mansfield, 2012) [5]. Measurement of canine pancreatic lipase immunoreactivity (cPLI) has become an important diagnostic tool owing to its improved sensitivity and specificity for pancreatic inflammation (Cridge *et al.*, 2021; Abhijith *et al.*, 2025) [3, 1]. Abdominal ultrasonography is widely used and may reveal pancreatic enlargement, hypoechoogenicity, surrounding hyperechoic mesentery, and abdominal effusion in affected dogs (Cridge *et al.*, 2021) [3].

Therapeutic management of severe pancreatitis is primarily supportive and includes aggressive fluid therapy, analgesia, antiemetic therapy, nutritional support, and correction of electrolyte and acid-base disturbances (Lim *et al.*, 2024) [4]. Early enteral nutrition and effective pain management have been shown to improve clinical outcomes and reduce systemic complications in canine pancreatitis (Mansfield and Beths, 2015) [6]. Despite advances in veterinary critical care medicine, severe acute pancreatitis continues to carry a guarded prognosis because of its potential to induce systemic inflammatory responses and multiorgan dysfunction (Mansfield, 2012) [5].

The present case report describes the clinical presentation, diagnostic findings, therapeutic management, and outcome of severe acute pancreatitis in a Beagle dog presented to the veterinary teaching hospital.

Case history and observations

A five-year-old intact male Beagle dog weighing 14.2 kg was presented with a history of repeated episodes of vomiting, anorexia, lethargy, abdominal discomfort, and progressive weakness of three days duration. The owner reported reduced feed intake and reluctance to move since the previous two days. Occasional episodes of diarrhea and excessive salivation were also noticed. The dog had reportedly consumed fatty table scraps prior to the onset of clinical signs. Vaccination and deworming status were up to date, and no previous history of major systemic illness was reported.

On general clinical examination, the animal appeared dull, depressed, and moderately dehydrated. Rectal temperature was elevated (103.8°F), heart rate was 148 beats/minute, and respiratory rate was 42 breaths/minute. Capillary refill time was prolonged (>3 seconds), indicating compromised peripheral perfusion. Mucous membranes were congested and tacky. The dog exhibited a characteristic praying posture intermittently and showed marked abdominal pain on cranial abdominal palpation. Mild abdominal distension and guarding response were also observed. Peripheral pulse quality was weak, suggestive of circulatory compromise.

Based on clinical examination, severe inflammatory gastrointestinal disease with systemic involvement was suspected. Differential diagnoses considered included acute pancreatitis, gastrointestinal obstruction, septic peritonitis, acute gastroenteritis, hepatobiliary disease, and toxin-induced gastroenteropathy.

Blood samples were collected for complete blood count and serum biochemical analysis. Hematological examination revealed marked leukocytosis with neutrophilia and mild hemoconcentration suggestive of acute inflammatory response and dehydration. Mild thrombocytopenia was also observed. Serum biochemical analysis demonstrated elevated serum Alanine Aminotransferase (ALT), Alkaline Phosphatase

(ALP), Blood Urea Nitrogen (BUN), and serum creatinine values. Hyperglycemia and electrolyte imbalances including mild hyponatremia and hypokalemia were also evident. Although serum amylase and lipase activities lack complete specificity for pancreatitis in dogs, marked elevation along with compatible clinical findings supported the presumptive diagnosis. Serum biochemical analysis additionally revealed hypocalcemia (7.3 mg/dL), which was considered suggestive of fat saponification and systemic inflammatory involvement associated with severe acute pancreatic inflammation.

The case was presumptively diagnosed as severe acute pancreatitis based on compatible clinical signs, hematobiochemical alterations, elevated serum amylase and lipase activities. The animal was immediately hospitalized for intensive monitoring and critical care management. Continuous monitoring of rectal temperature, heart rate, respiratory rate, hydration status, urine output, capillary refill time, blood glucose, and electrolyte status was undertaken throughout the treatment period.

Table 1: Hematological parameters before and after treatment

Parameter	Reference range	Day 1 (Pre-treatment)	Day 5 (Post-treatment)
Hemoglobin (g/dL)	11.9-18.9	17.2	14.8
Packed Cell Volume (%)	35-57	58	46
Total Erythrocyte Count ($\times 10^6/\mu\text{L}$)	4.95-7.87	8.9	7.1
Total Leukocyte Count ($\times 10^3/\mu\text{L}$)	5-14.1	24.6	12.8
Neutrophils (%)	58-85	89	72
Lymphocytes (%)	8-21	8	21
Platelet Count ($\times 10^3/\mu\text{L}$)	211-621	165	278
Blood Glucose (mg/dL)	76-119	168	108

Reference: Hematological and serum biochemical reference ranges were adapted from the MSD Veterinary Manual

Table 2: Serum biochemical alterations before and after treatment

Parameter	Reference range	Day 1 (Pre-treatment)	Day 5 (Post-treatment)
Alanine Aminotransferase (ALT) (U/L)	10-109	286	124
Alkaline Phosphatase (ALP) (U/L)	1-114	342	168
Blood Urea Nitrogen (BUN) (mg/dL)	8-28	48	22
Creatinine (mg/dL)	0.5-1.7	2.1	1.1
Total Protein (g/dL)	5.4-7.5	8.3	6.9
Albumin (g/dL)	2.3-3.1	2.1	3.0
Sodium (mEq/L)	142-152	132	146
Potassium (mEq/L)	3.9-5.1	3.1	4.4
Chloride (mEq/L)	110-124	96	108
Serum Amylase (U/L)	226-1063	2650	1320
Serum Lipase (U/L)	200-1800	4125	1765
Serum Calcium (mg/dL)	9.1-11.7	7.3	8.7

Reference: Hematological and serum biochemical reference ranges were adapted from the MSD Veterinary Manual

The hematological findings revealed marked leukocytosis with neutrophilia indicating severe inflammatory response, while elevated packed cell volume suggested dehydration and hemoconcentration. Altered renal parameters and electrolyte imbalance indicated systemic involvement secondary to severe pancreatic inflammation. Progressive normalization of hematobiochemical parameters following therapy indicated favorable clinical response to treatment.

Treatment and discussion

The dog was immediately hospitalized and intensive supportive therapy was initiated considering the severity of pancreatic inflammation and systemic compromise.

Intravenous fluid therapy using Ringer's lactate was administered at approximately 82 mL/hr based on AAHA fluid therapy guidelines after calculation of dehydration deficit, maintenance requirement, and ongoing gastrointestinal losses. The patient exhibited approximately 5% dehydration at presentation. Restoration of circulating volume and pancreatic perfusion was considered essential to minimize pancreatic ischemia and progression of inflammation.

Pantoprazole was administered intravenously at a dose rate of 1 mg/kg once daily to reduce gastric acid secretion and prevent stress-related gastric mucosal injury associated with severe vomiting and systemic illness. Proton pump inhibitors are commonly employed in canine pancreatitis to minimize gastrointestinal irritation and esophagitis secondary to repeated emesis.

Ceftriaxone was administered intravenously at 25 mg/kg twice daily for five days to minimize the risk of secondary bacterial infection and bacterial translocation associated with severe gastrointestinal inflammation. Pain management was achieved using buprenorphine administered intravenously at 0.02 mg/kg twice daily. Acute pancreatitis is recognized as an intensely painful condition due to pancreatic inflammation, capsular distension, and peripancreatic tissue involvement. Adequate analgesia is therefore considered an essential therapeutic component in affected dogs. Buprenorphine, a partial μ -opioid receptor agonist, provides effective visceral analgesia with minimal cardiovascular depression and has been widely recommended in management of acute pancreatitis.

Maropitant citrate was administered subcutaneously at 1.1 mg/kg once daily to control persistent vomiting and nausea. Maropitant acts as a neurokinin-1 receptor antagonist by blocking substance P within the emetic center and has been reported to provide effective antiemetic and mild visceral analgesic effects in dogs with pancreatitis.

Vitamin B12 supplementation was administered intramuscularly at a dose rate of 1 ml/10 kg body weight as supportive therapy. Cobalamin supplementation may aid gastrointestinal mucosal recovery and improve metabolic function in dogs suffering from severe gastrointestinal and pancreatic disease.

N-acetyl Cysteine (NAC) was administered intravenously at 140 mg/kg diluted in normal saline solution to reduce oxidative stress associated with severe pancreatic inflammation. Oxidative stress and excessive production of reactive oxygen species play a major role in acinar cell injury and systemic inflammatory responses during acute pancreatitis. N-acetyl cysteine acts as a glutathione precursor and antioxidant, thereby helping to reduce cellular oxidative damage and inflammatory mediator activity.

Food and water were withheld during the initial 24 hours of hospitalization to provide pancreatic rest and reduce pancreatic stimulation during the acute phase of inflammation. Nutritional management is considered an important aspect of pancreatitis therapy because inappropriate feeding may aggravate pancreatic enzyme secretion during severe disease (Mansfield and Beths, 2015) [6]. On the second day of treatment, a low-fat gastrointestinal prescription diet was gradually introduced in small frequent feedings. The patient tolerated enteral nutrition without recurrence of vomiting, and the gastrointestinal diet was continued for three days. By the sixth day of treatment, the dog regained normal appetite and was gradually transitioned to routine maintenance feeding.

Appropriate nutritional management and gradual introduction of enteral feeding are important components of supportive therapy in canine pancreatic disorders (Simpson *et al.*, 2001) [9].

The prognosis of canine pancreatic disorders varies depending on severity of pancreatic injury, extent of systemic involvement, and response to supportive therapy (Westermarck *et al.*, 2005) [13].

Analgesic therapy is considered essential in canine pancreatitis because abdominal pain is a major clinical component of pancreatic inflammation. Buprenorphine, a partial μ -opioid receptor agonist, provides effective visceral analgesia and is commonly recommended for management of mild to moderate pain associated with acute pancreatitis in dogs (Steiner and Williams, 1999; Mansfield and Beths, 2015) [10, 6]. Although routine antimicrobial therapy is not universally recommended in uncomplicated pancreatitis, antibiotics may be considered in severe cases with systemic inflammatory response, suspected bacterial translocation, or risk of secondary infection (Mansfield, 2012; Lim *et al.*, 2024) [5, 4].

Oxidative stress and excessive generation of reactive oxygen species contribute significantly to pancreatic acinar cell injury during acute pancreatitis. N-acetyl Cysteine (NAC), a glutathione precursor with antioxidant properties, has been reported to reduce oxidative damage and inflammatory mediator activity associated with pancreatic inflammation (Ramudo and Manso, 2010) [8].

Hypocalcemia observed in the present case may be attributed to fat saponification resulting from pancreatic fat necrosis and systemic inflammatory response. Decreased serum calcium concentration has been reported in severe canine pancreatitis and is often associated with increased disease severity and systemic involvement. Similar hematobiochemical alterations including leukocytosis and elevated hepatic enzymes have been previously reported in dogs with acute pancreatitis (Mansfield, 2012) [5].

Continuous monitoring of rectal temperature, pulse rate, respiratory rate, hydration status, abdominal pain, urine output, appetite, and hematobiochemical parameters was carried out throughout hospitalization. Marked clinical improvement was observed progressively following initiation of therapy. Vomiting subsided within 48 hours, hydration status improved, and abdominal discomfort significantly reduced. Repeat hematological and serum biochemical examination performed on the fifth day of treatment revealed marked reduction in leukocytosis, normalization of renal parameters, improvement in electrolyte balance, and considerable reduction in pancreatic enzyme levels, indicating favorable therapeutic response.

Acute pancreatitis in dogs is associated with variable prognosis depending on severity of pancreatic injury and degree of systemic involvement (Mansfield, 2012) [5]. Severe pancreatic inflammation results in activation of inflammatory cytokines, vascular endothelial injury, altered capillary permeability, and oxidative stress leading to systemic complications including shock, disseminated intravascular coagulation, and multiorgan dysfunction (Watson, 2015) [12]. Early aggressive fluid therapy, adequate analgesia, antioxidant therapy, antiemetic support, and nutritional management therefore remain essential for successful clinical recovery.

The favorable outcome observed in the present case highlights the importance of early diagnosis, intensive supportive care, appropriate nutritional management, and continuous monitoring in successful management of severe acute pancreatitis in dogs. The favorable response to aggressive fluid therapy and antiemetic management observed in the present case is consistent with previous reports.

Diagnosis of acute pancreatitis in dogs frequently relies on a combination of compatible history, clinical signs, hematobiochemical abnormalities, and response to therapy,

particularly when advanced diagnostic modalities are unavailable. In the present case, recurrent vomiting, abdominal pain, inflammatory leukogram, elevated serum amylase and lipase activities, dehydration, and favorable response to supportive treatment strongly supported a presumptive diagnosis of acute pancreatitis.

Conclusion

Severe acute pancreatitis in dogs can rapidly progress to a life-threatening condition if not recognized and managed promptly. The present case highlights the importance of early clinical assessment, hematobiochemical evaluation, and continuous monitoring in the presumptive diagnosis and successful management of acute pancreatitis in dogs. Although definitive confirmation using abdominal ultrasonography and canine pancreatic lipase immunoreactivity (cPLI) assay was not performed, compatible clinical signs, inflammatory hematobiochemical alterations, elevated serum amylase and lipase activities, hypocalcemia, and favorable therapeutic response strongly supported the diagnosis. Aggressive fluid therapy, effective analgesia, antiemetic therapy, antioxidant supplementation, and appropriate nutritional management played a crucial role in successful clinical recovery of the affected Beagle dog. Early intensive supportive care and timely therapeutic intervention remain essential to improve prognosis and minimize systemic complications associated with severe canine pancreatitis.

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