Short Communication

DIETARY FAT-INDUCED ACUTE PANCREATITIS IN A DOG: DIAGNOSTIC APPROACH AND MANAGEMENT

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ABSTRACT

Acute pancreatitis in dogs, characterized by sudden pancreatic inflammation and autodigestion, most commonly affects middle-aged to older patients. It typically results in abdominal pain, inappetence, and vomiting. While the underlying cause is often idiopathic, consumption of high-fat diets is a well-recognized trigger. A oneyear-old male Shih Tzu was presented with acute abdominal signs, including anorexia, vomiting, and cranial abdominal pain, following four months on a high-fat paneer-based diet. While abdominal radiography was unremarkable, ultrasonography revealed a thickened, hypoechoic right pancreatic lobe with surrounding hyperechoic mesenteric fat, suggestive of acute pancreatitis. Routine haematological and biochemical parameters, including serum amylase and lipase, were within normal limits; however, the serum canine-specific pancreatic lipase (Spec cPL) concentration was significantly elevated at 410 µg/L, confirming the diagnosis. Supportive management and dietary correction to a low-fat prescription diet resulted in complete clinical resolution by the seventh day. Follow-up Spec cPL concentration normalized to 55.9 µg/L within twenty days, and no recurrence was observed over a one-year period. This case highlights dietary fat as a key trigger and underscores the importance of prompt diagnosis using Spec cPL and ultrasonography, alongside targeted nutritional management for successful recovery.

Key words: abdominal pain, high fat diet, pancreatitis and canine specific pancreatic lipase

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INTRODUCTION

Acute pancreatitis, a common yet diagnostically challenging exocrine pancreatic disorder in dogs, most commonly affects middle-aged to older patients and is characterized by sudden inflammation leading to variable local and systemic manifestations (Xenoulis and Steiner,

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2008). This condition presents with an acute onset of clinical signs which may rapidly progress; severe cases can trigger systemic complications, significant morbidity, and mortality. Distinguishing clinically between acute and chronic forms remains difficult and often requires histopathological confirmation (Lim et al., 2024). Disease severity ranges from mild, self-limiting potentially presentations severe, to fatal systemic inflammatory responses. While the precise etiology often remains idiopathic, several predisposing factors are recognized, including dietary indiscretion, consumption of high-fat meals, specific medications, endocrine disorders, and breed susceptibilities (Watson, 2015).

The ingestion of high-fat or inappropriate human foods is a well-documented risk (Simpson, 2015). Sudden dietary intake of rich, fatty substances can overwhelm pancreatic enzymatic regulation. This dysregulation can lead to premature activation of digestive enzymes within the pancreas itself, resulting in autodigestion, inflammation, and consequent systemic effects (Williams *et al.*, 2016).

This report details a case of acute pancreatitis in a young Shih Tzu secondary to an unconventional, paneer-based diet, describing its clinical presentation, diagnostic evaluation, and successful therapeutic management.

CASE PRESENTATION

A one-year-old male Shih Tzu was presented with a history of anorexia and

vomiting for the past two days and signs of acute abdominal pain. The patient had been primarily maintained on a paneer-based diet for the preceding four months. Clinical examination revealed cranial abdominal pain, tachycardia (heart rate: 140 bpm), dehydration (~7%), and hyperaemic mucous membranes. Rectal temperature (102.3°F) and respiratory rate (22 breaths per minute) were within normal limits. By the third day of the disease onset, diarrhoea with melena was reported.

Abdominal radiography performed rule out other differentials (e.g., gastrointestinal obstruction, gastroenteritis, gastric dilatation and volvulus) revealed significant abnormalities. Due to the inconclusive radiographic findings and persistent clinical signs, abdominal ultrasound was performed. This revealed a thickened (1.2 cm), irregular, and hypoechoic right pancreatic lobe, exceeding the normal reference range (6.3–9.9 mm) (Fig. 1). Surrounding areas revealed hyperechoic mesenteric fat, a thickened duodenal wall with intraluminal speckles (Fig 2). The remaining abdominal organs appeared normal. These ultrasonographic findings supported a strong suspicion of acute pancreatitis.

Hematological and routine serum biochemical parameters were within normal limits. Serum amylase and lipase activities were unremarkable; however, the serum canine-specific pancreatic lipase (Spec cPL) concentration was found to be significantly elevated at 410 $\mu g/L$, confirming the diagnosis of pancreatitis.

The patient was managed with nil per os (NPO) status for three days. Supportive care included intravenous Ringer's lactate fluid therapy (60 ml/kg administered twice daily), ondansetron (0.2 mg/kg IV twice daily) for antiemesis, pantoprazole (1 mg/kg IV once daily) for gastric acid suppression, and butorphanol (0.3 mg/kg IV thrice daily) for analgesia. Gradual clinical improvement was noted by the third day, characterized by reduced vomiting and the passage of semi-solid faeces. A lowfat gastrointestinal prescription diet was then cautiously introduced and was well tolerated. The patient exhibited complete resolution of clinical signs by the seventh day of hospitalization.

Approximately twenty days after initial presentation, the serum canine-specific pancreatic lipase (Spec cPL) concentration was re-evaluated and found to be within the normal reference range i.e.,55.9 μ g/L. No recurrence of clinical signs was observed during a one-year follow-up period.

DISCUSSION

Acute pancreatitis, a serious inflammatory disorder of the exocrine pancreas in dogs, manifests with sudden and often severe clinical signs. The clinical presentation of pancreatitis in dogs is highly variable, ranging from mild, self-limiting illness to severe, life-threatening disease, and is consistently marked by non-specific signs (Xenoulis, 2015).

Vomiting and abdominal pain represent the earliest and most common,

albeit non-specific, clinical signs of acute pancreatitis. Dogs with severe disease typically present with an acute onset of anorexia, weakness, vomiting, diarrhoea, and/or abdominal pain (Lim *et al.*, 2024).

Elevated serum amylase and lipase were considered pancreas-specific markers; however, their diagnostic value is limited as elevations can occur secondary to reduced renal clearance and other non-pancreatic disorders (Watson, 2015). While complete blood count, serum biochemistry, and urinalysis findings are non-specific for pancreatitis, these tests remain essential for excluding differential diagnoses and evaluating the patient's systemic status (Xenoulis and Steiner, 2012).

radiography Abdominal was unremarkable in this case. Radiographs diagnostic limited utility pancreatitis in dogs and cats, as findings are frequently normal or reveal only nonspecific alterations. Potential changes, such as increased soft tissue opacity or decreased serosal detail in the cranial right abdomen, displacement of abdominal organs, gaseous dilation of adjacent bowel loops, and abdominal effusion, lack specificity and can be associated with numerous other abdominal conditions (Ferreri et al., 2003).

Regarding diagnostic imaging, abdominal ultrasonography revealed key findings: pancreatic enlargement (specifically a thickened, hypoechoic right pancreatic lobe), hyperechoic surrounding mesenteric fat (indicating steatitis), and duodenal wall thickening with intraluminal

speckles (consistent with duodenitis). Characteristic ultrasonographic findings in canine pancreatitis include pancreatic enlargement, hypoechogenicity (potentially indicating necrosis or oedema), irregular pancreatic margins, increased echogenicity of the peripancreatic fat (due to necrosis/inflammation), and occasionally pancreatic duct dilation or abdominal effusion (Hecht and Henry, 2007).

Immunoassays measuring canine pancreatic lipase immunoreactivity (cPLI) offer significant advantages over traditional enzymatic tests due to their superior sensitivity and specificity (Watson, 2015). Consequently, the diagnosis of acute pancreatitis in dogs typically relies on a combination of clinical signs, imaging findings, and specific pancreatic enzyme assays. Serum canine pancreatic lipase (Spec cPL) has emerged as a highly sensitive and specific diagnostic marker, becoming widely accepted in small animal practice (Cridge et al., 2021). Although histopathology remains the definitive diagnostic standard, its invasive nature and potential to exacerbate pancreatic injury preclude its routine clinical use (French et al., 2019). This limitation has increased reliance on non-invasive tools, including thorough clinical evaluation, cPL assays, and abdominal ultrasonography.

The management of acute pancreatitis in dogs is primarily supportive and symptomatic. Key principles include identifying and addressing the underlying

cause, aggressive fluid therapy, managing complications, administering antiemetics and analgesics, and providing appropriate nutritional support (Leppaniemi et al., 2020). In this case, pancreatitis was directly attributed to a prolonged high-fat diet (paneer), and clinical signs resolved completely following dietary correction. Dietary factors, particularly the ingestion of high-fat or inappropriate human foods, represent significant and preventable triggers for pancreatitis (Simpson, 2015). Excessive fat intake can overwhelm pancreatic enzymatic regulation, leading to premature intracellular activation proteases. autodigestion of the gland, and subsequent local and systemic inflammation (Williams et al., 2016). Early enteral nutrition utilizing supports fat-restricted diets recoverv by minimizing pancreatic stimulation and reducing the risk of gastrointestinal complications (Wingert et al., 2021).

CONCLUSION

This case demonstrates acute pancreatitis triggered by a high-fat paneer diet. Diagnosis was confirmed through characteristic ultrasonographic findings combined with elevated serum Spec cPL. Supportive medical therapy and immediate transition to a fat-restricted prescription diet achieved complete clinical resolution, with normalized Spec cPL levels and sustained recovery.

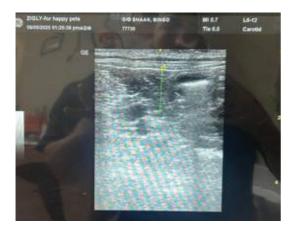


Fig .1. Thickened, irregular, hypoechoic right pancreatic lobe, measuring 1.2 cm in thickness with surrounding hyperechoic mesenteric fat

REFERENCES

Cridge, H., MacLeod, A.G., Pachtinger, G.E., Mackin, A.J., Sullivant, A.M., Thomason, J.M., and Steiner, J.M. (2021). Spec cPL for the diagnosis of pancreatitis in dogs: a systematic review. *Journal of Veterinary Internal Medicine*, **35**(3): 1337–1351.

Ferreri, J.A., Hardam, E., Kimmel, S.E., Saunders, H.M., Van Winkle, T.J., Drobatz, K.J., and Washabau, R.J. (2003). Clinical differentiation of acute necrotizing from chronic nonsuppurative pancreatitis in cats: 63 cases (1996-2001). Journal of the American Veterinary Medical Association, 223(4): 469–474.

French, J.M., Twedt, D.C., Rao, S., and Marolf, A.J. (2019). Computed tomographic angiography and

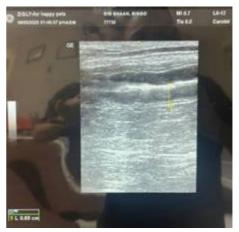


Fig .2. Duodenal wall thickening with intraluminal speckles.

ultrasonography in the diagnosis and evaluation of acute pancreatitis in dogs. *Journal of veterinary internal medicine*, **33**(1): 79–88.

Hecht, S. and Henry, G. (2007). Sonographic evaluation of the normal and abnormal pancreas. Clinical Techniques in Small Animal Practice, 22: 115-121

Leppaniemi, A., Tolonen, M., Tarasconi, A., Segovia-Lohse, H., Gamberini, E., Kirkpatrick, A.W., Ball, C.G., Parry, N., Sartelli, M., Wolbrink, D., van Goor, H., Baiocchi, G., Ansaloni, L., Biffl, W., Coccolini, F., Di Saverio, S., Kluger, Y., Moore, E. E., and Catena, F. (2020). Executive summary: WSES Guidelines for the management of severe acute pancreatitis. *The Journal of Trauma and Acute Care Surgery*, **88**(6): 888–890.

- Lim, S.Y., Cridge, H., Twedt, D.C., Ohta, H., Nuruki, T., and Steiner, J.M. (2024). Management of acute-onset pancreatitis in dogs: a narrative Review. *Journal of the American Veterinary Medical Association*, **262**(9): 1231–1240.
- Simpson, K.W. (2015). Pancreatitis and other disorders of the exocrine pancreas. In E. Cote (Ed.), *Clinical Veterinary Advisor: Dogs and Cats* (pp. 748–755). Saunders Elsevier.
- Watson, P.J. (2015). Pancreatitis in dogs and cats: definitions and pathophysiology. *Journal of Small Animal Practice*, **56**(1): 3–12.
- Williams, T.L., Mealey, K.L., and Milovancev, M. (2016). Canine pancreatitis. *Compendium: Continuing Education for Veterinarians*, **38**(2): E1–E10.

- Wingert, A.M., Murray, O.A., Lulich, J.P., Hoelmer, A.M., Merkel, L.K., and Furrow, E. (2021). Efficacy of medical dissolution for suspected struvite cystoliths in dogs. *Journal of Veterinary Internal Medicine*, **35**(5):2287–2295.
- Xenoulis, P.G. (2015). Diagnosis of pancreatitis in dogs and cats. *Journal of Small Animal Practice*, **56**(1), 13–26.
- Xenoulis, P.G., and Steiner, J.M. (2008). Canine pancreatitis. *Journal of Veterinary Internal Medicine*, 22(5):1159–1170.
- Xenoulis, P.G., and Steiner, J.M. (2012). Diagnostic approach to pancreatitis in dogs and cats. *Journal of Veterinary Emergency and Critical Care*, **22**(2), 77–86.