B-5367
[1-4]

SHORT COMMUNICATION Indian Journal of Anim

Management of Acute Pancreatitis and Kidney Injury in a Dog

K. Mohanambal[1], D. Sumathi[1], R. Ravi[1], S. Sivaraman[1],
K.K. Ponnu Swamy[1], M. Elavarasan[1], P. Preethi[1]

10.18805/IJAR.B-5367

ABSTRACT

Concurrent acute pancreatitis and acute kidney injury represent significant pathological conditions in dogs, presenting challenges in diagnosis and management. In human medicine this is interconnected and complex co morbid condition. Renal impairment consequently leads to fluid and electrolyte imbalance, in turn causes metabolic disturbances that may predispose to pancreatitis in dogs. Identification of underlying etiology and predisposing factors prevents further recurrence in affected individuals. The critical interplay between AP and AKI needs comprehensive management procedure. This article poses a case study of an eight-year-old Dalmatian cross dog presenting with severe respiratory distress, jaundice and gastrointestinal symptoms. Laboratory findings revealed elevated markers of pancreas, liver and kidney injury. Prompt intervention with fluid therapy, antibiotics and supportive care led to significant clinical improvement. Subsequent re-evaluation demonstrated resolution of pancreatic inflammation and restoration of renal function. This case underscores the importance of early recognition, aggressive intervention and comprehensive management in achieving successful outcomes in dogs with concurrent pancreatitis and acute kidney injury in dogs.

Key words: Acute kidney injury, Acute pancreatitis, Butorphanol, Jaundice, Respiratory distress.

Concurrent acute pancreatitis (AP) and acute kidney injury (AKI) represent significant pathological conditions poses heightened complexities in their management and necessitates a thorough understanding of their pathogenesis. AKI is defined by a rapid reduction in renal function, triggered by many factors like sepsis, low cardiac output, hypovolemia, major surgeries, urinary obstruction and ingestion of toxic drugs. Owing to impaired elimination of nitrogenous waste products resulted accumulation of serum urea and creatinine levels in serum, causes anorexia, weakness and vomiting along normal or reduced urine output in the affected individuals (Beker *et al.,* 2018 and Hwang *et al.,* 2019). AP is a well known inflammatory disorder reported in dogs characterized by early activation of pancreatic enzymes, often resulting from infection, underlying medication, fatty diet, hyperlipidemia, trauma and genetic predisposition. In extreme cases it can lead to pancreatic necrosis along with systemic inflammation. The affected animal exhibit signs of severe abdominal pain and some cases typical praying posture. Assessment of pancreatic enzymes and imaging of pancreas and interpretation confirms of pancreatitis. According to the complication and duration AP is classified as mild, moderate and severe (Banks *et al.,* 2013). Diagnosis of AP is challenging in dogs because of absence of consistent and universally accepted gold standard test. Traditionally, histopathology was considered as valid and unique to differentiate AP from chronic pancreatitis (Xenoulis, 2015). But in this case acquisition of sample is tough and affected dogs were poor anaesthetic candidates. So recently diagnosis of pancreatitis relies on a combined assessment of patient history, physical examination, evaluation of pancreatic lipase levels and ultrasonographic or advanced imaging techniques. However, these approaches primarily recognize severe cases of AP (Enoe *et al.,* 2000 and Branscum *et al.,* 2005). Concurrent AKI exacerbates the risk of mortality in critically ill patients due to hemodynamic changes, microcirculatory dysfunction, systemic inflammation-related injury and increased intra-abdominal pressure. Diagnosis necessitates the evaluation of multifactorial influences contributing to the onset of both conditions, elucidating potential triggers, predisposing factors and underlying pathophysiological pathways. Through the comprehensive investigation intricate interplay between pancreatitis and kidney injury in canines, in turn facilitating the development of more refined preventive, diagnostic and therapeutic approaches. Despite the recognized connection between AP and AKI in human medicine, specific clinical studies investigating this relationship in dogs are lacking. However, given the parallel pathophysiological mechanisms and clinical observations

[1]Department of Veterinary Clinical Medicine, Veterinary College and Research Institute, Namakkal-637 002, Tamil Nadu, Veterinary and Animal Sciences University, Chennai, Tamil Nadu, India.

Corresponding Author: K. Mohanambal, Department of Veterinary Clinical Medicine, Veterinary College and Research Institute, Namakkal-637 002, Tamil Nadu, Veterinary and Animal Sciences University, Chennai, Tamil Nadu, India.
Email:mohanambalmvsc@gmail.com

How to cite this article: Mohanambal, K., Sumathi, D., Ravi, R., Sivaraman, S., Swamy, K.K.P., Elavarasan, M. and Preethi, P. (2024). Management of Acute Pancreatitis and Kidney Injury in a Dog. Indian Journal of Animal Research. DOI: 10.18805/IJAR.B-5367.

Submitted: 28-03-2024 **Accepted:** 10-06-2024 **Online:** 23-07-2024

in both species, further exploration of this association in veterinary medicine could provide valuable insights for diagnosis and management strategies. The aim of this study is to record the clinical manifestations, diagnostic modalitites, hematobiochemical alterations and comprehensive treatment strategies for concurrent occurrence of AP and AKI and to pave a way to future researchers to plan for precise diagnosis and effective treatment.

Case History, Clinical observation and Laboratory Investigation

An eight-year-old Dalmatian crossbreed dog was presented to the small animal medicine outpatient unit with severe respiratory distress, accompanied by an elevated body temperature of 39.2°C. Detailed owner history revealed previous treatment with inj. *Lasix* and inj. Dexamethasone for suspected gastritis with respiratory distress along with Salbutamol nebulization. Despite the respiratory distress, the dog had normal heart rate and rhythm. The dog appeared to be in good general condition with a placid expression. However, upon examination, signs of jaundice were noted, including icteric mucous membranes, icteric corneas and icteric changes in the skin (Fig 1). The dog had not been eating for the past four days and showed reluctance to move. Additionally, there were intermittent episodes of vomiting, although there was no diarrhoea. No abnormalities were observed in lymph nodes and ectoparasites such as ticks liceand fleas were absent. It was planned to evaluate haematology, serum biochemistry, pancreatic enzymes, radiography and ultrasonography. Haematobiochemical changes in the presented dog were indicative of severe illness and abnormal liver and kidney function markers (Table 1). Haemoglobin (Hb) level was low at 9.5 g/dL, while the packed cell volume (PCV) was 28%, indicating possible anaemia and decreased red blood cell volume. High white blood cell count was suggesting infection or inflammation (54.49×10^3/µL). Elevated liver enzyme levels may indicates heapatic steatosis. Changes in serum creatinine and blood urea nitrogen (BUN) levels were owing to systemic inflammation and hypoperfusion in turn suggestive of acute kidney injury. Elevated levels of lipase and amylase suggest possible pancreatitis and damage to pancreatic tissue. Radiography was insignificant, but ultrasonography evidenced enlarged pancreas with irregular margin (Fig 2), other abdominal organs showed normal study. These findings highlight a complex medical situation requiring thorough critical care management, follow up and treatment.

Overall, laboratory tests revealed leucocytosis with mild anaemia in haematology and serum biochemistry indicated elevated levels of creatinine, total and indirect bilirubin, pancreatic lipase and amylase. Radiographic study did not reveal any abnormalities. However, abdominal ultrasonography revealed an enlarged left lobe of the pancreas with irregular margins, indicative of pancreatic inflammation and oedema (Fig 2 and 3).

The dog was promptly treated with intravenous fluids (inj. Ringer's lactate and inj. dextrose normal saline each @ 10ml/kg, IV, once in 8 hours), inj. metronidazole(@ 10 mg/kg, IV, once in 12 hours), inj. butorphanol (@ 0.5 mg/kg, IV, once in 8 hours), inj. ondensedran (@10 ml/kg, IV, once in 12 hours), inj. pantaprazole (@ 1 mg/kg, IV, once a day), inj.furosemide (@ 2 mg/kg, IV, once in 12 hours). Oral probiotic supplements, phosphate binder (Tab. Sevelamer 400) and oral food was withheld for three days to allow the pancreas to rest. The therapy initiated showed improvement by the second day and the owner was advised to reintroduce water and liquid diet. Treatment was continued for five days,

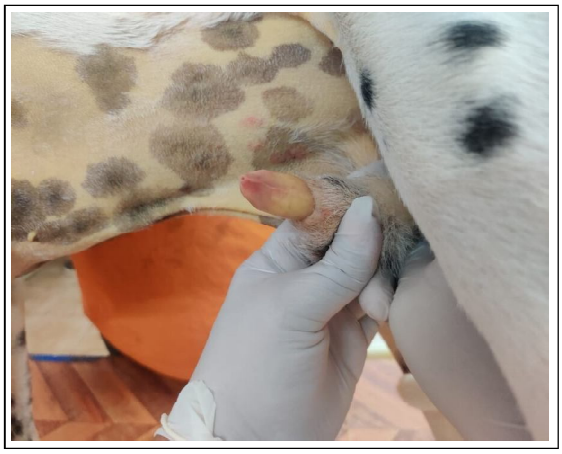

Fig 1: Icteric changes in the skin in a dog with pancreatitis.

Table 1: Haematobiochemical alterations in the affected dogs.

Parameters	Day 0	Day 5	Day 7
Haematology			
Hb (g/dL)	9.5	15.1	16
PCV (%)	28	42	44
RBC ($\times 10^6$/µL)	4.2	6.3	6.5
WBC ($\times 10^3$/µL)	54.49	40.51	35.12
Neutrophils (%)	82	76	73
Lymphocytes (%)	04	19	22
Monocytes (%)	04	05	05
Platelets ($\times 10^3$/µL)	205	415	546
Serum biochemistry			
Total protein (g/dL)	7.8	7.8	7.3
Albumin (g/dL)	2.9	3.0	3.2
AST (U/L)	229	55	44
ALT (U/L)	114	48	41
SAP (U/L)	1224	612	118
Total bilirubin (mg/dL)	11.8	1.0	0.8
Direct bilirubin (mg/dL)	8.6	0.4	0.3
BUN (mg/dL)	118	25	20
Creatinine (mg/dL)	9.4	1.5	1.0
Glucose (mg/dL)	62	34	77
Pancreatic enzymes			
Serum lipase (U/L)	1095	545	316
Serum amylase (U/L)	3024	1244	612

during which there was significant improvement in the condition of the dog. Subsequent haemato-biochemistry showed drastic improvement and oral medication (tab. metronidazole, tab. enrofloxacin) was continued for a total of nine days. After completion of therapy, ultrasonography and blood samples were collected again for re-evaluation, which revealed normal pancreatic tissue and nearly normal blood profile. Over the course of the observation period, significant improvements were observed in various haematological and biochemical parameters. Haemoglobin (Hb) levels showed a remarkable increase from Day 0 (9.5 g/dL) to Day 7 (16 g/dL), indicating reversal of anaemic condition and enhanced oxygen-carrying capacity to peripheral tissue. Similarly, the platelet count exhibited a notable rise, suggesting improved hemostatic function. Furthermore, liver enzymes including AST, ALTand ALP demonstrated significant decreases from Day 0 to Day 7, indicative of reduced hepatocellular damage. Bilirubin levels exhibited a marked decrease over the same period, reflecting improved liver function and bile metabolism. Additionally, blood urea nitrogen (BUN) and creatinine levels declined substantially from Day 0 to Day 7, suggesting improved renal function. Moreover, serum lipase and amylase levels showed progressive decreases, indicating resolution of pancreatic inflammation and injury. These findings collectively suggest a positive response to treatment and overall improvement in the clinical condition of the dog over the observed timeframe. Overall, the dog responded well to the treatment regimen prescribed, leading to a successful resolution of the acute pancreatitis and associated complications.

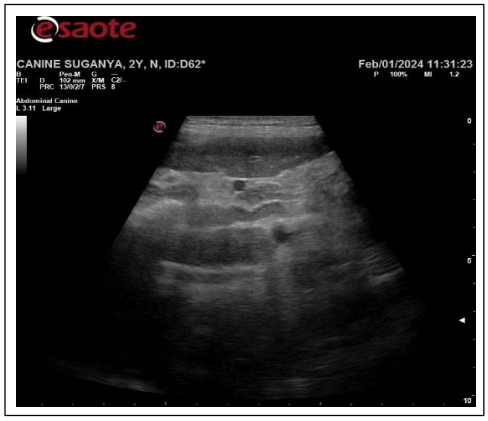

Fig 2: Abdominal ultrasonography revealed an enlarged left lobe of the pancreas.

Acute pancreatitis and acute kidney injury often occur together and are linked to poor outcomes in both human and veterinary medicine. In the current study acute pancreatitis diagnosis was diagnosed on the presence of clinical indications such as abdominal pain, diarrhoea, vomiting or decreased appetite, with enlarged pancreas during abdominal ultrasound, coupled with an abnormal serum pancreatic lipase and amylase (Gori *et al.,*2019). Similarly, AKI was diagnosed based on IRIS guidelines, the dog had normal urine output. Whereas, Gori *et al.* (2019) was reported worse prognosis in dogs with oligo-anuria. Acute pancreatitis can lead to AKI in dogs through mechanisms such as hypovolemia, cytokine-induced ischemia, inflammation and oxidative stress. The dog with pancreatitis associated with acute kidney injury was survived, even it had higher ALP (1224 U/L), total bilirubin (11.8 mg/dL) and direct bilirubin (8.6 mg/dL) levels. This is contradictory to the report of Gori *et al.* (2019) diagnosed, pancreatitis in 22% of the dogs with acute kidney injury which had no significant difference in its occurrence between survivors and non-survivors. However, activities of liver enzymes (ALP, ALT, AST, GGT) and bilirubin concentration were significantly higher in non-survivors, indicating potential complications such as pancreatitis and liver injury, which could contribute to a worse outcome. In the current study mild anaemia was reported which is in accordance with Rimer *et al.* (2022) was observed anaemia in dogs with acute kidney injury (AKI) admitted to referral

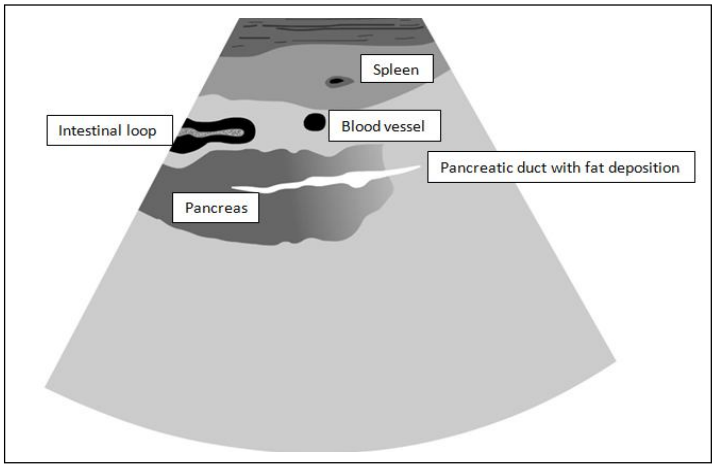

Fig 3: The schematic representation of inflamed pancreas.

centres. In the present study even though the presented dog had high creatinine level (9.4 mg/dL) it had survived, the same was documented by Segev *et al.* (2008) and Gori *et al.* (2019) that serum creatinine concentration at presentation was not linked to mortality, suggesting that prognosis should not be solely based on serum creatinine levels at the time of admission. If we treat the etiology for acute kidney injury promptly the nature of kidney injury can be reversed. Eatroff *et al.* (2012) and Dunaevich *et al.* (2020) reported additional complications like liver injury may worsen the outcome. Additionally, the number of organs affected by the diseases was positively associated with the case fatality rate, emphasizing the importance of considering multi-organ involvement in prognostic assessments (Segev *et al.,* 2008).

CONCLUSION

Concurrent acute pancreatitis and acute kidney injury represent serious pathological conditions in dogs, necessitating a thorough understanding of their pathogenesis and targeted therapeutic interventions. This case report highlights the efficacy of aggressive fluid resuscitation, analgesia and supportive care in achieving successful resolution of pancreatic inflammation and restoration of renal function. Early recognition, prompt intervention and close monitoring are paramount in managing these complex conditions and minimizing associated morbidity and mortality in canine patients. Continued detailed research and keen clinical observations are essential to further refine diagnostic and therapeutic strategies for optimal management of concurrent pancreatitis and kidney injury in dogs.

Conflict of interest

The authors don't have any conflict of interest for the manuscript.

REFERENCES

Banks, P.A., Bollen, T.L., Dervenis, C., Gooszen, H.G., Johnson, C.D., Sarr, M.G., Tsiotos, G.G. and Vege, S.S. (2013). Classification of acute pancreatitis-2012: Revision of the Atlanta classification and definitions by international consensus. Gut. 62: 102-111.

Beker, B.M., Corleto, M.G., Fieiras, C. and Musso, C.G. (2018). Novel acute kidney injury biomarkers: Their characteristics, utility and concerns. Int. Urol. Nephrol. 50: 705-713.

Branscum, A.J., Gardner, I.A. and Johnson, W.O. (2005). Estimation of diagnostic-test sensitivity and specificity through Bayesian modeling. Preventive Veterinary Medicine. 68(2-4):145-63.

Dunaevich, A., Chen, H., Musseri, D., Kuzi, S., Mazaki Tovi, M., Aroch, I. and Segev, G. (2020). Acute on chronic kidney disease in dogs: Etiology, clinical and clinicopathologic findings, prognostic markers and survival. J. Vet. Intern. Med. 34: 2507-2515.

Eatroff, A.E., Langston, C.E., Chalhoub, S., Poeppel, K. and Mitelberg, E. (2012). Long-term outcome of cats and dogs with acute kidney injury treated with intermittent hemodialysis: 135 cases (1997-2010). J. Am. Vet. Med. Assoc. 241: 1471-1478.

Enoe, C., Georgiadis, M.P. and Johnson, W.O. (2000). Estimation of sensitivity and specificity of diagnostic tests and disease prevalence when the true disease state is unknown. Preventive Veterinary Medicine. 45(1-2): 61-81.

Gori, E., Lippi, I., Guidi, G., Perondi, F., Pierini, A. and Marchetti, V. (2019). Acute pancreatitis and acute kidney injury in dogs. The Veterinary Journal. 245: 77-81.

Hwang, S., Park. H., Kim, Y., Kang, D., Ku, H. S, Cho, J., Lee, J. E., Huh, W., Guallar, E., Suh, G. Y. and Jang, H. R. (2019). Changes in acute kidney injury epidemiology in critically ill patients: A population-based cohort study in korea. Ann. Intensive Care. 9:1-9.

Rimer, D., Chen, H., Bar Nathan, M. and Segev, G. (2022). Acute kidney injury in dogs: Etiology, clinical and clinico pathologic findings, prognostic markers and outcome. Journal of Veterinary Internal Medicine. 36(2): 609-618.

Segev, G., Kass, P.H., Francey, T. and Cowgill, L.D. (2008). A novel clinical scoring system for outcome prediction in dogs with acute kidney injury managed by hemodialysis. Journal of Veterinary Internal Medicine. 22(2): 301-308.

Xenoulis, P.G. (2015). Diagnosis of pancreatitis in dogs and cats. Journal of Small Animal Practice. 56(1): 13-26.