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Case Report

Management of acute pancreatitis induced acute kidney injury by intermittent hemodialysis in a labrador retriever dog

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Abstract

A 08-year-old male Labrador Retriever dog with history of anorexia, lethargy, severe vomiting, abdominal pain, weight loss, polyuria, polydipsia and melena from last 4 days was presented to hospital. Initial physical assessment revealed congested mucus membrane, with other vital parameters within normal physiological range. Hematological examination revealed neutrophilic leukocytosis with mild left shift. Biochemistry revealed Blood urea nitrogen (BUN)- 171 mg/dL, Creatinine-6.5 mg/dL, Lipase-1869 u/L and cPLI-414.8ng/mL. Abdominal ultrasound demonstrated hyperechoic cortex with a mild loss of cortico-medullary differentiation (CMD) in both kidneys, suggesting AKI. Blood gas analysis indicated metabolic acidosis with compensatory alkalosis. Initially, aggressive fluid therapy and other conservative treatment were administered for two days to mitigate the AKI. However, due to a further increase in creatinine levels to 7.2 mg/dL, IHD was initiated to manage the AKI. After four sessions of IHD, the dog recovered uneventfully. This case highlights the significance of early intervention with IHD in dogs afflicted with pancreatitis-induced AKI who remain unresponsive to conservative treatment. Azotemia serves as a negative prognostic indicator and necessitates prompt correction. Intermittent hemodialysis (IHD) demonstrated efficacy in restoring renal function and stabilizing metabolic parameters. The timely utilization of IHD can be life-saving and enhance long-term outcomes in canine AKI.

Keywords: Acute kidney injury, Blood gas analysis, Intermittent hemodialysis, Pancreatitis

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INTRODUCTION

Acute pancreatitis (AP) is a sudden inflammation of the exocrine pancreas characterized by severe vomiting, anorexia, abdominal pain, and diarrhea. (Mansfield et al., 2008; Xenoulis, 2015). Pancreatitis is a highly destructive and life-threatening condition that affects approximately 80,000 individuals annually in the United States. (Liu et al., 2025), whereas it is considered as the most common disorder of exocrine pancreas in dogs and cats (Bandivadekar and Swamy, 2020). Acute kidney injury (AKI) involves acute disturbances in renal function leading to accumulation of uremic waste and acid-base disturbances in the body (Ross, 2011). AKI and AP are the prevalent comorbidities in human medicine (Petejova and Martinek, 2013) whereas in veterinary medicine AKI and AP are associated with an increased risk of mortality (Gori et al., 2019).

Acute pancreatitis can induce acute kidney injury (AKI) due to inflammation, cytokine-induced ischemia, hypovolemia, and oxidative stress (Mansfield et al., 2008). Intra-abdominal hypertension, increased capillary permeability, and nephrotoxic pancreatic enzymes additionally compromise renal perfusion and directly damage the tubular epithelium, thereby perpetuating ischemia–reperfusion injury and progression of AKI in the setting of AP (Barton et al., 2020). In the absence of histopathology, a comprehensive clinical history, physical examination, abdominal ultrasound, and canine pancreatic lipase test are currently employed to establish a diagnosis of AP in dogs (Xenoulis, 2015). In dogs with acute pancreatitis (AP), acute kidney injury (AKI) has been reported in approximately 31.5% of cases, with severity ranging from Grade I to IV according to IRIS criteria (Gori et al., 2020). Analogously to human counterparts, hospitalized dogs diagnosed with AKI induced by AP exhibit a significantly elevated mortality risk compared to non-AKI patients (Zhou et al., 2015).

Although the etiology of AP induced AKI in dogs is well-established, only a limited number of studies (Bendeler et al., 2023) have been published regarding effective management of AP-induced AKI with advanced renal replacement therapies such as hemodialysis. This highlights the need for further research to elucidate the clinical presentation, robust diagnostic methods, and optimal management strategies for AP-induced AKI in dogs to enhance clinical outcomes.

HISTORY CLINICAL DIAGNOSIS AND FINDING

An eight-year-old intact male weighing 31kg Labrador Retriever explosive detection dog, assigned to the Railway Protection Force (RPF) of Northern Indian Railways, was brought to the emergency and critical care ward of Multispecialty Veterinary Hospital (MSVH), Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana. The hospital registration number of the dog was 14306. A comprehensive medical history assessment revealed the presence of anorexia, lethargy, recurrent vomiting, abdominal pain, weight loss, polyuria, polydipsia, and melena over the past four days. Initial physical assessment revealed body condition score (BCS)-2.5 (on 5-point scale), congested mucus membrane, dull mentation, dehydration 6-8%, rectal temperature 101.40F, heart rate-70 beats per minute, respiration rate-28 breaths per minute with normal superficial lymph nodes and systolic blood pressure (SBP) I40 mmHg (Doppler Vet BP mano medical, France).

Hematological examination revealed hemoglobin (Hb) 15.3 g/dL, total leucocyte count (TLC) 35,000 along with neutrophilic leukocytosis with mild left shift. Biochemistry revealed blood urea nitrogen (BUN)- 171 mg/dL, creatinine -6.5 mg/dL, sodium (Na)-147 mEq/L, potassium (K)- 4.1 mEq/L, chloride (Cl)- 97mEq/L, phosphorus (P)- 10.1mg/dL, lipase-1869 u/L and canine pancreatic lipase immunoreactivity assay (cPLI)- 414.8ng/mL (Dot-ELISA kit (Spec cPL®, IDEXX Laboratories, Inc., Westbrook, Maine, USA) (Reference Interval (RI) >400 ng/ml is consistent with pancreatitis). Routine urine analysis revealed urine specific gravity

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of 1.050, urine protein creatinine ratio (UPCR)- 0.3 and urine pH- 5. Abdominal ultrasound demonstrated a hypoechogenic pancreas with an increased size and surrounding echogenic diameter exceeding 1.2 cm (Figure 1). Also, both kidneys exhibited hyperechoic cortices with a mild loss of cortico-medullary differentiation (CMD), indicating AKI. Based on the laboratory findings, the dog was classified as having non-oliguric AKI, corresponding to IRIS Stage IV.

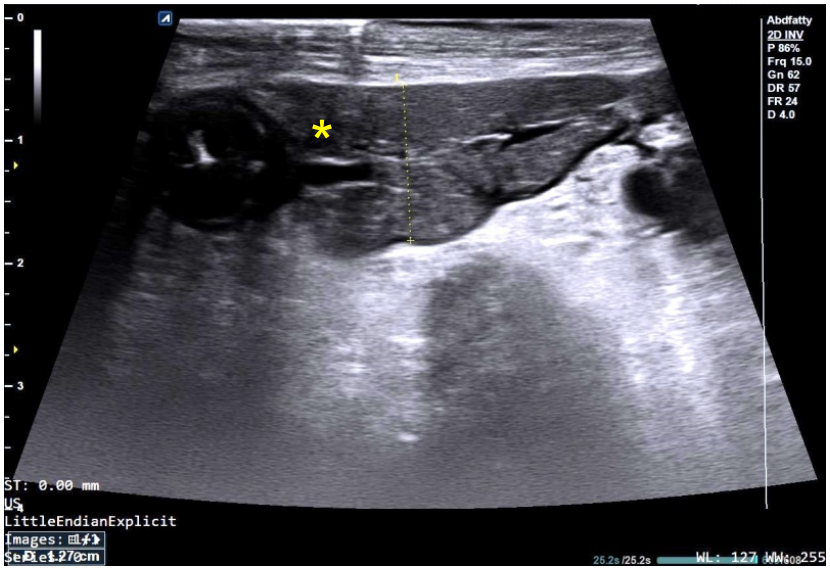


Figure 1 Abdominal ultrasound of pancreas (*) with increased pancreatic diameter and surrounding hyperechogenic fat

CASE MANAGEMENT

Initially, to mitigate the AKI, aggressive fluid therapy and other conservative treatment measures, including diuretics and antiemetics, were administered for duration of two days. However, due to a subsequent increase in the dog's creatinine level to 7.2 mg/dL, the animal was referred to the dialysis unit, where IHD was selected as the appropriate management strategy for the AKI.

Following aseptic preparation of the catheterization site, a dedicated 19 cm, 11.5 Fr double-lumen temporary dialysis catheter (DLC) was inserted into the left jugular vein to establish vascular access. The catheter tip was meticulously positioned at the confluence of the cranial vena cava and the right atrium (Figure 2) to ensure optimal and uninterrupted blood flow. Intermittent hemodialysis (IHD) was subsequently initiated using a Fresenius Medical Care 4008S dialysis platform. To establish extracorporeal circulation, a blood tubing (AV-SETFMC PAED-R, Fresenius Medical Care Germany) with an inner diameter of 6.4 mm and a filling volume of 117 ml was utilized in this study. The low-flux dialyzer (FX5) employed in the present case was manufactured by Fresenius Medical Care, Germany, and utilized a helixone membrane with surface area of 1.0 m2. The low-flux membrane possessed a small pore size, enabling the removal of toxins with low molecular weight. In session I, the dialysis session duration and blood pump speed (Qb) were kept @ 2 mL/kg/min for 2 hours; session II @ 5 mL/ kg/ min for 3 hours and III, IV @ 7 mL/kg/min for 4 hours. Dialysate flow rate (Qd) was kept at 500mL/min to obtain a well-balanced dialysis efficiency whereas, ultrafiltration (UF) rate was kept at 100mL during all sessions During ongoing dialysis, continuous monitoring of patient vital signs (rectal temperature, electrocardiogram, and arterial oxygen saturation) was achieved using a multi-parameter monitor (Phillips G30E, USA).

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Blood pressure monitoring was conducted every 15 minutes using a Doppler sphygmomanometer (Doppler Vet BP mano medical, France). Coagulation was monitored manually using activated clotting time (ACT) assessment which was maintained between 160-200 seconds. The results guided heparin dose adjustments to maintain adequate anticoagulation throughout dialysis.

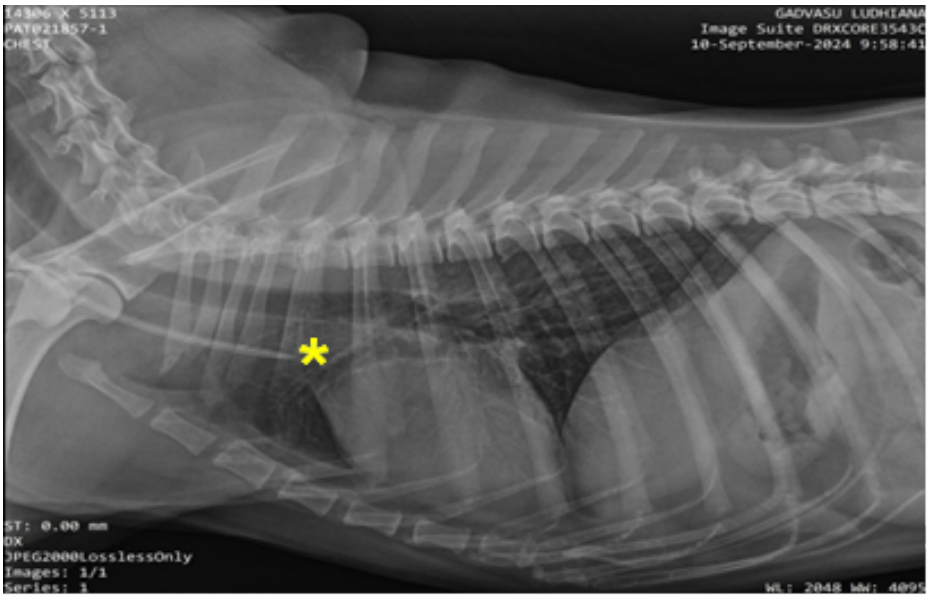


Figure 2 Thoracic radiograph showing correct placement of double lumen dialysis catheter tip at the junction of cranial vena cava and right atrium (*).

The tabulated data delineate the physiological and biochemical responses of a canine subject undergoing four sequential sessions of IHD, with session durations progressively increasing from 120 to 280 minutes (Table 1). A marked amelioration in renal function was observed, which was evidenced by a substantial decline in BUN from 171 to 18 mg/dL and serum creatinine from 7.4 to 2.1 mg/dL. Serum phosphorus levels decreased from 11.2 to 3.7 mg/dL, indicating effective phosphate clearance. Electrolyte homeostasis was largely maintained, with serum sodium and chloride levels remaining within physiological range. However, a transient reduction in potassium levels was noted post-IHD. Hematological parameters exhibited variability with Hb concentration reducing from 15.3 to 11.1 g/dL, and reduction in platelet count, potentially reflecting hemodilution or dialysis-associated losses. Liver enzyme activities remained relatively stable throughout the treatment course with minor fluctuations in total protein and albumin levels (Table 1). Notably, pancreatic enzyme markers, such as lipase and cPLI, exhibited substantial decreases, indicating a potential reduction in pancreatic stress or inflammation. Venous blood gas analysis was conducted both pre- and post-IHD across four sessions to monitor metabolic disturbances during ongoing dialysis.

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Table 1 Comparison of pre-and post-hematobiochemical parameters in dog undergoing intermittent hemodialysis (IHD)

Parameter	Session-I		Session-II		Session-III		Session-IV	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Hemoglobin (g/dL) (RI-12-18)	15.3	15.1	13.8	14	12.9	11.7	11.5	11.1
TLC (ul) (RI-6000-17000)	35000	32000	30600	30400	26700	29300	20300	25000
PCV (%)(RI-37-55)	44	42	38.5	39.5	35.1	32.6	32.3	32.4
Platelet (10^3/ul) (RI-200-500)	373	119	146	110	178	76	287	180
ALT (U/L) (RI- 8.2-57)	62	57	69	68	65	64	67	65
GGT (U/L) (RI-1-10)	26	15	22	20	24	20	22	20
Total Protein (g/dL) (RI-5.4-7.5)	8.3	8.5	8.2	7.7	7.9	6.3	6.7	7.3
Albumin (g/dL)(RI-2.6-3.3)	3.9	3.3	4.1	2.8	2.9	3.1	2.9	3.2
BUN (mg/dL) (RI-8-28)	171	66	99	34	57	25	39	18
Creatinine (mg/dL) (RI-0.5-1.7)	7.4	4.4	5.1	2.9	4.7	2.7	4.4	2.1
Phosphorus (mg/dL) (RI-2.9-5.3)	11.2	4.1	6.9	5.6	6.2	4.2	5.2	3.7
Sodium (mEq/L) (RI-142-152)	147	145	139	141	141	135	140	132
Potassium (mEq/L) (RI-3.9-5.1)	4.1	2.1	2.5	2.6	2.6	2.5	3.1	2.9
Chloride (mEq/L) (RI-110-124)	97	99	95	99	97	99	98	99
Glucose (mg/dL) (RI-62-108)	105	98	102	95	99	89	97	89
Calcium (mg/dL) (RI-8.7-11.8)	10.7	12	11.4	10.9	11.7	11.3	11.6	11.8
Lipase (U/L) (RI-<500)	1869	-	-	-	-	-	-	470
cPLI (ng/ml) (RI-<400)	414.8	-	-	-	-	-	-	123.9

Session-I = Day-1 (Session time: 120 minute)
Session-II = Day-2 (Session time: 180 minutes)
Session-III = Day-4 (Session time: 240 minutes)
Session-IV = Day-6 (Session time: 280 minutes)

The venous blood gas and electrolyte analysis revealed notable improvements in acid-base balance (Table 2). A gradual increase in pH was observed post-IHD in each session, indicating correction of acidosis. Bicarbonate (HCO_3^-) levels increased significantly post-dialysis, particularly in Sessions I and III (from 18.3 to 24.8 mmol/L and 27.6 to 40.7 mmol/L, respectively), reflecting improved buffering capacity. Base excess (BE) and total CO_2 (TCO_2) showed corresponding rises. While pCO2 (Partial pressure of carbon dioxide) values remained relatively stable, cSO2 (oxygen content of blood) showed fluctuations, with some post-IHD reductions. The anion gap (AGap) and potassium-adjusted anion gap (AGapk) remained within acceptable limits, with a trend toward normalization by the fourth session. Overall, IHD effectively contributed to metabolic stabilization of the dog.

During dialysis sessions, the dog received pantoprazole (1 mg/kg BW) intravenously once-daily, maropitant (1 mg/kg BW) subcutaneously once-daily, ampicillin (22 mg/kg BW) intravenously twice-daily, and enrofloxacin (5 mg/kg BW) intramuscularly twice-daily. Post-dialysis medications included L-carnitine (20 mg/kg BW) intravenously once-daily and an essential amino acid infusion (inj. Nephrosteril 7%, Fresenius Kabi, Thailand) @ 250 mL intravenously once-daily.

Following four sessions of IHD, the dog exhibited significant clinical improvement, with a return to normal appetite. At the six-month follow-up post-IHD, the dog remained clinically active, with no recurrence of clinical signs. Blood parameters, including creatinine, lipase, and cPLI, remained within normal limits, indicating sustained recovery and stable renal and pancreatic function.

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Table 2 Comparison of pre-and post- venous blood gas analysis in dog undergoing intermittent hemodialysis (IHD)

Parameter	Session-I		Session-II		Session-III		Session-IV	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
pH (RI-7.31-7.42)	7.290	7.450	7.451	7.485	7.481	7.421	7.482	7.401
pCO_2 (mmHg) (RI-29-42)	37	35.6	38.2	40.2	37.1	37	36.8	42.3
$cHCO_3$ (mmol/L) (RI-17-24)	18.3	24.8	26.6	30.3	27.6	24.1	40.7	26.2
BE (ecf) (mmol/L) (RI- -2 - -3)	4.9	0.8	2.6	6.9	4.2	-0.4	4.1	1.5
cSO_2 (%) (RI-94-98)	67.9	55.5	71.1	39	57.1	74.6	79.9	47.4
TCO_2 (mmol/L) (RI-22-29)	28.3	24.7	26.5	30.4	27.6	23.9	27.5	26.4
Agap (mmol/L) (RI-7-15)	14	20	14	14	11	11	12	5
AGapk (mmol/L) (RI-10-20)	16	22	16	16	14	14	14	9

Session-I = Day-1 (Session time: 120 minute)
Session-II = Day-2 (Session time: 180 minutes)
Session-III = Day-4 (Session time: 240 minutes)
Session-IV = Day-6 (Session time: 280 minutes)

DISCUSSION

Acute kidney injury (AKI) in dogs is a critical condition often associated with high morbidity and mortality, especially when secondary to systemic diseases such as AP (Rimer et al., 2021). In the present case, the dog exhibited classical signs of AP with concurrent azotemia, as indicated by elevated levels of serum creatinine, BUN and pancreatic biomarkers (lipase and cPLI). Azotemia is considered a negative prognostic indicator in dogs with AP, and its presence is associated with an increased risk of mortality (Papa et al., 2007; Mansfield et al., 2008; Marchetti et al., 2023).

Despite initial management with aggressive fluid therapy and supportive care, renal parameters continued to deteriorate, necessitating extracorporeal renal replacement therapy (IHD) (Segev et al., 2024). Dogs with AKI that fail to respond to conventional medical treatment have poor outcomes if not treated promptly with advanced renal replacement modalities (Ross, 2011). Intermittent hemodialysis (IHD) has emerged as an effective therapeutic option for managing severe azotemia, correcting electrolyte imbalances, and stabilizing acid-base disturbances in dogs with AKI (Singh et al., 2025). The hemodialysis prescription in this case was designed following the recent IRIS best practice consensus guidelines (Segev et al., 2024), which emphasize gradual adaptation of treatment intensity to optimize solute clearance while minimizing adverse effects. The dialysis duration and blood flow rates were progressively increased over sessions to allow safe hemodynamic adjustment and effective toxin removal. Ultrafiltration (UF) rates were kept conservative, and close clinical and biochemical monitoring guided subsequent session planning. This individualized and guideline-based approach resulted in stable hemodynamics, absence of dialysis disequilibrium, and steady improvement in renal parameters, reflecting the positive clinical impact of a carefully titrated hemodialysis prescription. We choose i-IHD in this case which involves tailoring the initial IHD prescription according to residual kidney function. After 4 sessions the dog recovered uneventfully without any further need of performing dialysis.

In this case, IHD led to marked improvements in renal function, with a substantial reduction in serum creatinine alongside normalization of phosphorus levels (Singh et al., 2024). In our study, significant reductions in post-dialysis levels of BUN, creatinine, and phosphorus were consistent findings. This is a common occurrence in both human and animal patients undergoing IHD (Cowgill and Francey, 2012). BUN, creatinine and phosphorus are commonly used uremic parameters to monitor the effectiveness of dialysis sessions (Malchert et al., 2017;

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Singh et al., 2024). The recent IRIS best-practice consensus guidelines for dogs and cats emphasized that IHD is an advanced adjunct to standard care, reserved mainly for severe AKI or stage IV CKD, and that the number and frequency of sessions should be individualized based on the degree of azotemia, fluid overload, electrolyte and acid–base derangements, and the likelihood of renal recovery (Segev et al., 2024). These changes reflect the efficiency of IHD in removing nitrogenous waste products and controlling hyperphosphatemia, a common complication in AKI (Singh et al., 2025). The venous blood gas parameters underwent substantial alterations during IHD, with a significant correction of acid-base equilibrium (Yamamoto et al., 2017). The most prominent alterations included pH fluctuations, bicarbonate loss, and pCO2 depletion (Wieliczko and Małyszko, 2022). Metabolic acidosis was the primary alteration, accompanied by partial respiratory compensation (Rezende et al., 2017). The mean pH value transitioned from mild to moderate acidity during the pre-dialysis session and gradually approached normal during the post-dialysis session (Goutham et al., 2019). Session I demonstrated normalization of pH, indicating effective correction of systemic acidosis (Lugon et al., 2018). Subsequent sessions observed a shift in pH to the higher limit, accompanied by a moderate increase in bicarbonate levels (Wieliczko and Małyszko, 2022). This pattern corresponds to the anticipated outcome of hemodialysis, which entails the removal of uremic toxins and restoration of pH equilibrium through the provision of bicarbonate-rich dialysate (Cuadrado et al., 2022). Anion gap, which typically ranges from 12 to 24, was found to be at a lower borderline range, suggesting the possibility of non-anion gap metabolic acidosis (NAGMA) (Kunchur et al., 2014). This condition indicates bicarbonate loss, renal tubular acidosis, or hyperchloremic/uremic acidosis. During the four IHD sessions pCO2 remained within normal range. There was moderate hypoxemia during the post-dialysis period, which may be linked to fluid shifts and the reaction of the blood with the artificial dialyzer membrane (Uzun et al., 2022). Although within the normal range, oxygen saturation (CSO_2) showed a declining trend throughout the four sessions which could be due to alterations in hemoglobin-O2 affinity as blood cells pass through the extracorporeal circuit and changes in fluid dynamics during IHD (Zhang et al., 2018). Overall, IHD demonstrated a consistent and progressive rectification of metabolic acidosis and enhanced acid-base parameters without significantly altering the anion gap, thereby underscoring its efficacy and safety in addressing uremic acid-base imbalances in canines (de Azevedo et al., 2025). Similar study in human hemodialysis patients (Marano et al., 2017) has demonstrated most significant impact on the PaO2 and SaO2 variables. Likewise, in another study involving human (Rindaha et al.,2015) CKD patients undergoing dialysis, metabolic acidosis was the most common acid-base disturbance, evident in 22 (41.5 %) patients along with respiratory alkalosis in five (9.4 %) patients and respiratory acidosis in two (3.8 %) patients. Renal failure in dogs and humans primarily concerns acid-base disequilibrium. Similar findings were observed in a recent study (de Azevedo et al., 2025), where dogs undergoing IHD in by-pass mode showed increases in blood pH, bicarbonate levels, and base excess.

Fluctuations in hematobiochemical values, including a decline in hemoglobin and variable platelet counts, along with reduced total protein and albumin levels may be attributed to hemodilution or minor losses associated with dialysis circuits (Langston et al., 2017; Singh et al., 2024). Importantly, serum pancreatic enzyme concentrations (lipase and cPLI) significantly decreased post-IHD, suggesting concurrent resolution of pancreatic inflammation or reduced pancreatic enzyme release, supporting the hypothesis that effective management of AKI can positively impact comorbidities like AP (Takada et al., 2018).

The comprehensive long-term follow-up demonstrated complete clinical recovery, with stable renal and pancreatic biomarkers even six months after treatment. This outcome underscores the longevity of therapeutic response following IHD in well-selected cases. This result is consistent with previous studies

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that have reported favorable prognoses and improved quality of life in dogs that survive the initial critical phase of AKI with the assistance of renal replacement therapy (Cowgill, 2012).

In summary, this case highlights the clinical efficacy of IHD in managing severe AKI secondary to AP in dogs. It underscores the paramount importance

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS

Sachin, Singh R, and Singh RS contributed to the conceptualization and data curation of the case report, with Singh R and Park HM also playing key roles in the formal analysis. The investigation was primarily conducted by Sachin and Singh R, while resources were provided by Singh RS and Park HM. Supervision of the case was carried out by Singh R, Singh RS, and Park HM, with validation undertaken by Singh R and Park HM. Visualization and drafting of the original manuscript were performed by Sachin, while all authors, including Sachin, Singh R, Singh RS, and Park HM, contributed to the critical review and editing of the final manuscript.

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