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# Diabetic ketoacidosis complicated by chronic kidney disease in a domestic shorthair cat

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**ABSTRACT:** Diabetic ketoacidosis (DKA) is a life-threatening metabolic complication secondary to diabetes mellitus (DM), a common endocrine disorder in cats. This condition is characterized by severe dehydration, electrolyte imbalance, and metabolic acidosis resulting from insulin deficiency and excessive ketone production. A 7-year-old castrated male cat presented with dyspnea, vomiting, marked dehydration, and hypothermia. Laboratory findings revealed severe hyperglycemia, glucosuria, ketonuria, azotemia, and elevated hepatic enzyme levels, confirming DKA complicated by chronic kidney disease. The cat received intensive therapy, including oxygen supplementation, intravenous fluids with potassium, insulin administration, antibiotics, and supportive care. Although transient improvement in electrolyte levels was achieved, glycemic control remained unstable, and the patient succumbed after three days of hospitalization.

**Keywords:**
feline, diabetes mellitus, diabetes ketoacidosis, electrolyte imbalance, insulin

## ■ INTRODUCTION

Diabetes mellitus (DM) is one of the most common endocrine disorders in cats, with increasing prevalence over the past decade, and obesity as a major predisposing factor (Hoenig, 2014). According to Rudloff (2017), while cats with DM may remain stable for months, diabetic ketoacidosis (DKA) can rapidly develop because of illness, stress, or increased metabolic demand.

DKA is a severe DM complication caused by insulin deficiency, counter-regulatory hormones, and dehydration (Rudloff, 2017). Animals show dehydration, electrolyte imbalances, and metabolic acidosis, requiring therapy. DKA occurs with disorders such as pancreatitis, urinary infection, hyperadrenocorticism, or renal insufficiency (Panciera, 2012). Animals with diabetes and DKA exhibit lethargy, vomiting, weakness, and weight loss. Diagnosis requires ketonuria detection in animals with diabetes. Prognosis depends on the underlying disease (Rand, 2013).

Successful management of feline DKA requires prompt recognition, fluid resuscitation, correction of electrolyte imbalances, and administration of insulin to restore glycemic control. Concurrent disorders, such as pancreatitis or infections, must be treated, as they often precipitate DKA (Rudloff, 2017). This report describes the presentation and management of diabetic ketoacidosis complicated by chronic kidney disease (CKD) in a cat.

## ■ CASE

**Signalment and Anamnesis**: A 7-year-old castrated male domestic shorthair cat, weighing 6.1 kg with BCS 9/9, presented to St. Angel Animal Medical Center, Malaysia. The blind, fully vaccinated cat showed hematemesis, reduced appetite, and respiratory difficulty for two days. There was no endocrine or renal disease. **Physical Examination**: The cat showed depression and obtundation, prolonged capillary refill, a temperature of 36°C, dehydration 8%, and breathing rapid. The heart and abdomen were normal. **Diagnostic Tests**: Hyperglycemia, DKA, and elevated blood urea nitrogen (BUN) and creatinine levels, indicating renal compromise (Table 1). Urinalysis showed ketones and glucose (Table 2). **Diagnosis**: Diabetic ketoacidosis (DKA) complicated by chronic kidney disease (CKD). **Differential Diagnosis**: Acute necrotizing pancreatitis causes insulin resistance and ketoacidosis. Sepsis mimics DKA. **Prognosis**: Concurrent renal dysfunction and delayed stabilization lead to poor outcomes. **Treatment and Management**: Oxygen, fluids, and potassium were administered. Insulin reduced glucose levels. Warming and antibiotics were administered. Despite hydration, glycemic control was poor. The cat died on day three.

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## ■ RESULTS AND DISCUSSION

The primary goals of managing feline diabetic ketoacidosis (DKA) are correcting fluid and electrolyte imbalances and restoring glycemic control through insulin therapy. Careful monitoring is essential to prevent a rapid decline in plasma osmolality and blood glucose and sodium levels (Table 3 and 4), which may cause neurological complications. Cats with critical signs, such as dyspnea, poor tissue perfusion, or circulatory collapse, require immediate intensive stabilization. Impaired perfusion in cats typically manifests as pale mucous membranes, prolonged capillary refill times, and hypotension (Little, 2015). Minimizing stress during treatment is crucial because it can exacerbate metabolic instability and hinder recovery.

The patient, Tuah, was admitted to St. Angel Animal Medical Center Malaysia with dyspnea and vomiting. Clinical evaluation revealed DKA with CKD and hepatic injury. Treatment included oxygen, intravenous fluids, azodyl (1 capsule BID), samilyn (1 tablet BID), omeprazole (1 mg/kg BW SID), maropitant (1 mg/kg BW SID), amoxicillin–clavulanate (15 mg/kg BW SID), and insulin (1 U when blood glucose exceeded 15 mmol/L).

Table 1. Haematology of a cat with diabetic ketoacidosis complicated by chronic kidney disease

| Parameter | Reference | Result |
|---|---|---|
| RBC (M/µL) | 6.54 – 12.20 | 6.38 |
| HCT (%) | 30.3 – 52.3 | **26.2** |
| HGB (g/dL) | 9.8 – 16.2 | 9.5 |
| WBC (K/µL) | 2.87 – 17.02 | **21.02** |
| GLU (mg/dL) | 71 – 159 | **>686** |
| CREA (mg/dL) | 0.8 – 2.4 | **too high** |
| BUN (mg/dL) | 16 – 36 | **101** |
| TP (g/dL) | 5.7 – 8.9 | 7.6 |
| ALT (U/L) | 12 –130 | **187** |
| ALKP (U/L) | 14 – 111 | 84 |

Note: RBC= red blood cell; HCT= hematocrit; HGB= hemoglobin; WBC= white blood cell; GLU= glucose; CREA= creatinine; BUN= blood urea nitrogen; TP= total protein; ALT= alanine aminotransferase; ALKP= alkaline phosphatase; blue=low value; red=high value.

Table 2. Urinalysis of a cat with diabetic ketoacidosis complicated by chronic kidney disease

| Parameter | Results |
|---|---|
| Colour | Dark Yellow |
| Clarity | Clear |
| Specific Gravity | 1.024 |
| pH | 6.5 |
| Prothrombin (mg/dL) | 30 |
| Glucose (mg/dL) | 1000 |
| Ketone (mg/dL) | 15 |
| Urobilinogen (mg/dL) | 12 |
| Bilirubin (mg/dL) | 3 |
| Blood (Ery/µL) | 50 |

Table 3. Electrolyte monitoring during hospitalization.

| Parameter | Reference | Day 1 | Day 2 | Day 3 |
|---|---|---|---|---|
| Na (mmol/L) | 150—165 | **147** | **147** | 159 |
| K (mmol/L) | 3.5—5.8 | **2.2** | 3.0 | 4.8 |
| Na/K (mmol/L) | 28.4—42.9 | **68** | **49** | 33 |
| Cl (mmol/L) | 112—129 | **99** | **103** | 116 |

Note: blue=low value; red=high value

Table 4. Blood glucose monitoring during hospitalization (mmol/l).

| Time | Day 1 | Day 2 | Day 3 |
|---|---|---|---|
| 03:00 | >15 | 14.1 | 22.2 |
| 07:00 | >15 | 20.3 | 24.9 |
| 11:00 | >15 | 10.0 | 25.8 |
| 15:00 | 22.7 | 17.6 | Deceased |
| 19:00 | 15.1 | 15.4 | Deceased |
| 23:00 | 7.6 | 18.4 | Deceased |

Fluid therapy is vital for DKA management, with cats showing 7–12% dehydration (Rand, 2013). Tuah received Lactated Ringer's solution (60 mL/kg/day) with potassium chloride (25 mEq/L). While 0.9% sodium chloride is the standard (Rudloff, 2017; Rand, 2013), Lactated Ringer's was chosen for alkalinizing effects. The electrolytes were monitored throughout (Table 3). Potassium supplementation is needed because potassium levels decline after insulin therapy (Rudloff, 2017). Insulin began 2 hours post-fluids using subcutaneous glargine (1 U/cat/12h if glucose >14–16 mmol/L) (Table 4), targeting 4 mmol/L/h reduction (Rand, 2013).

Renal-specific feeding was used as a nutritional sup-port. Early nutrition is vital, as anorexia increases catabolism in overweight cats. Low-carbohydrate diets are preferred (Rand, 2013). Despite electrolyte correction, Tuah's glycemic control remained unstable until death on day three. DKA prognosis depends on concurrent diseases, with 18–36% mortality in referral hospitals versus 100% survival in uncomplicated cases (Rand, 2013).

## ■ CONCLUSION

This case demonstrates the rapid progression of diabetic ketoacidosis in cats with chronic kidney disease. Early recognition, fluid therapy, and electrolyte correction are crucial for survival in feline DKA.

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