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Popular Article

Diabetic Ketoacidosis: A Critical Endocrine Emergency in Veterinary Practice

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Abstract:

Diabetic ketoacidosis (DKA) is a life-threatening metabolic complication of diabetes mellitus characterized by insulin deficiency, increased counter-regulatory hormone activity, hyperglycaemia, excessive ketone production and metabolic acidosis. Although diabetes mellitus is commonly encountered in small-animal practice, progression to ketoacidosis represents a medical emergency requiring rapid recognition and intensive management. The pathogenesis of DKA involves an imbalance between insulin and counter-regulatory hormones, including glucagon, cortisol, catecholamines, vasopressin and growth hormone. This hormonal disturbance promotes hyperglycaemia, lipolysis and hepatic ketogenesis, ultimately resulting in dehydration, electrolyte disturbances, hyperketonaemia and metabolic acidosis. Clinical signs may include anorexia, vomiting, polyuria, polydipsia, weakness, depression, dehydration, weight loss and altered respiratory pattern. Concurrent disorders such as pancreatitis, infections, renal disease and endocrinopathies may precipitate or worsen DKA and should be actively investigated. Diagnosis relies on assessment of hyperglycaemia, ketosis and acid-base status, supported by evaluation of electrolytes and other laboratory parameters. Treatment is centred on restoration of circulating volume, correction of electrolyte and acid-base abnormalities, suppression of ketogenesis with insulin therapy and identification and treatment of concurrent diseases or precipitating factors. Frequent monitoring of blood glucose, ketones, electrolytes and clinical status is essential during therapy. Advances in veterinary practice, including continuous glucose monitoring (CGM), are providing new opportunities for closer assessment of glucose trends during intensive diabetic management. Early diagnosis, systematic stabilization and vigilant monitoring can substantially improve the clinical outcome of veterinary patients with DKA.

Key words: Diabetic ketoacidosis, Diabetes mellitus, Metabolic acidosis, Hyperglycaemia, Insulin therapy

Introduction:

Diabetes mellitus is an important endocrine disorder encountered in dogs and cats and results from inadequate insulin activity and consequent abnormalities in glucose metabolism. While uncomplicated diabetes mellitus can often be managed successfully with appropriate insulin therapy, nutritional

management and regular monitoring, some patients develop diabetic ketoacidosis (DKA), a potentially fatal metabolic emergency. DKA develops secondary to insulin deficiency or defective insulin-mediated signalling and glucose transport (insulin resistance), resulting in inadequate glucose uptake into cells (Gant *et al.*, 2024). It develops when inadequate insulin activity occurs together with an increase in counter-regulatory hormones. The resulting hormonal imbalance promotes hepatic glucose production, decreases peripheral glucose utilisation and stimulates lipolysis and ketone-body formation. Ketone bodies are produced by the liver and are used as an energy source when glucose is not available. The two main ketone bodies are acetoacetate and β -hydroxybutyrate (β -HB), while acetone is the least abundant ketone body (Weingart *et al.*, 2012).

Diabetic ketoacidosis was defined as blood glucose >240 mg/dL, plasma pH < 7.3, and/or plasmatic bicarbonate 1.9 mmol/L) and/or ketonuria (Silva *et al.*, 2021). The accumulation of ketoacids, together with severe osmotic diuresis caused by hyperglycaemia, produces a complex clinical syndrome involving dehydration, hypovolaemia, electrolyte depletion, hyperosmolality and metabolic acidosis. High-anion-gap metabolic acidosis is the most frequently reported acid-base disturbance in dogs and cats affected with DKA (Cuddy *et al.*, 2025). The transition from diabetes mellitus to DKA may be associated with stressful events or concurrent disease. Infection, pancreatitis, hyperadrenocorticism and renal disease have been reported among concurrent conditions in diabetic patients, while certain medications may also contribute to metabolic decompensation. Clinically, affected animals may present with nonspecific signs such as anorexia, lethargy, weakness, vomiting, dehydration, polyuria, polydipsia and weight loss. Severe cases may develop characteristic changes in respiratory pattern. Consequently, a high index of suspicion is essential, particularly when a diabetic patient deteriorates rapidly or develops gastrointestinal and systemic signs.

DKA should therefore be approached not simply as severe hyperglycaemia, but as a multisystem metabolic emergency. Successful management requires simultaneous attention to fluid deficits, insulin deficiency, ketone production, electrolyte abnormalities, acid-base disturbances and the underlying precipitating disease. Advances in veterinary emergency and critical care have improved the diagnostic and therapeutic approach to DKA. Blood glucose measurement, ketone assessment, blood gas analysis and electrolyte monitoring allow clinicians to evaluate the major metabolic abnormalities and monitor their correction during treatment. More recently, continuous glucose monitoring (CGM) has emerged as a useful technology for obtaining frequent glucose measurements and identifying glucose trends, providing an additional tool for intensive diabetic management. Understanding the pathophysiology is fundamental to recognising these abnormalities and managing the patient safely. Early recognition, systematic stabilization and careful monitoring remain the cornerstones of successful management of this potentially fatal endocrine emergency.

Pathophysiology of Diabetic Ketoacidosis:

Diabetic ketoacidosis develops when insulin deficiency is accompanied by an increase in counter-regulatory hormones, particularly glucagon, cortisol, epinephrine and growth hormone (Hume *et al.*, 2006). This imbalance shifts the body from normal glucose utilisation towards excessive glucose production and fat breakdown. The resulting metabolic changes ultimately lead to hyperglycaemia, ketone accumulation, dehydration, electrolyte disturbances and metabolic acidosis.

- **Insulin deficiency and hyperglycaemia:**

Reduced insulin activity prevents adequate glucose uptake by peripheral tissues while promoting hepatic gluconeogenesis and glycogenolysis. As glucose continues to accumulate in the bloodstream, marked hyperglycaemia develops. The increased blood glucose concentration causes osmotic diuresis, resulting in excessive urinary loss of water and electrolytes and progressive dehydration.

- **Lipolysis and ketogenesis:**

Insulin normally suppresses fat breakdown. When its activity is inadequate, lipolysis increases and large amounts of free fatty acids are released into circulation. These fatty acids are taken up by the liver and used for ketone-body production. At the same time, increased glucagon activity promotes hepatic ketogenesis. When ketone production exceeds the body's ability to utilise and eliminate these compounds, hyperketonaemia develops.

- **Metabolic acidosis:**

The accumulation of ketoacids in the circulation overwhelms the body's buffering capacity, resulting in metabolic acidosis. Ketones are also excreted through the kidneys along with cations, further contributing to electrolyte losses. Thus, DKA is not simply a disorder of elevated blood glucose, but a complex metabolic disturbance involving glucose, lipid, fluid and electrolyte metabolism.

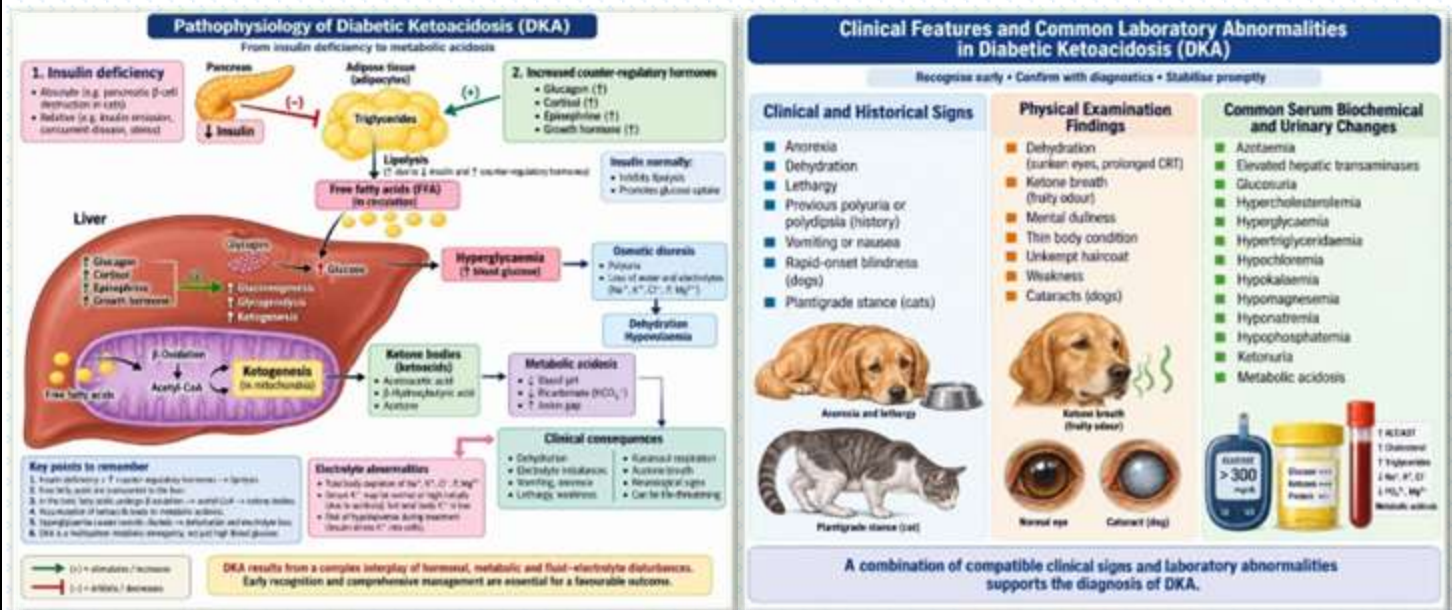
- **Dehydration and electrolyte disturbances:**

Hyperglycaemia-induced osmotic diuresis, together with vomiting and reduced water intake, produces substantial fluid and electrolyte losses. Sodium, potassium, phosphorus and magnesium may be affected. Importantly, total body potassium is depleted even when the initial serum potassium concentration appears normal because acidosis initially shifts potassium from cells into the extracellular space. During treatment, insulin administration and correction of acidosis can drive potassium back into cells, potentially causing a rapid decline in serum potassium.

Diagnostic Approach:

Diagnosis of DKA is based on the combination of compatible clinical signs, hyperglycaemia, ketosis and metabolic acidosis. Dogs and cats with DKA commonly present with anorexia, vomiting, lethargy, weakness, polyuria, polydipsia and dehydration. Physical examination may reveal ketone breath, mental dullness, poor body condition and an unkempt coat. Dogs may develop cataracts or sudden blindness,

while cats may occasionally show a plantigrade stance.



A sick diabetic animal showing anorexia, vomiting, dehydration or lethargy should be evaluated for DKA. Blood glucose, urinalysis, blood or urine ketones, electrolytes and blood-gas analysis are useful for confirming the diagnosis and assessing its severity. The standard method of detection of ketone bodies is the dipstick method, which detects semi quantitatively acetoacetate, but not β -hydroxybutyrate (β -HB). Blood β -hydroxybutyrate (BHB) measurement can detect ketosis earlier than urine ketone testing (Silva *et al.*, 2021).

Laboratory Abnormalities:

Typical laboratory abnormalities include hyperglycaemia, glucosuria, ketonuria or ketonemia, metabolic acidosis, azotaemia and electrolyte disturbances. Electrolyte disturbances such as hyponatraemia, hypochloreaemia, hypokalaemia, hypomagnesaemia and hypophosphatemia may also occur. Hypercholesterolaemia and hypertriglyceridemia can develop because of altered lipid metabolism associated with insulin deficiency (Cuddy *et al.*, 2025).

Treatment and Emergency Management:

DKA requires prompt stabilisation and hospitalisation, particularly in severely dehydrated, hypotensive, obtunded or markedly acidotic patients. Treatment focuses on restoring circulating volume, correcting electrolyte and acid-base abnormalities, suppressing ketogenesis with insulin and identifying the underlying precipitating disease. Common triggers include infections, pancreatitis, hypercortisolism and renal disease (Hurrell *et al.*, 2016)

• Insulin Therapy:

The treatment of DKA consists mainly of the administration of exogenous insulin either intravenously, in the form of continuous infusion or intramuscularly to interrupt lipolysis and the consequent formation of ketone bodies (Silva *et al.*, 2021). In DKA, insulin should be initiated after

initial fluid resuscitation and assessment of potassium status. Current AAHA guidance describes regular insulin protocols for both dogs and cats, with frequent glucose monitoring and adjustment according to the patient's response. Importantly, insulin therapy should continue even when blood glucose falls, with dextrose supplementation, when necessary, because the therapeutic goal is resolution of ketosis rather than simply lowering blood glucose.

• Fluid and Electrolyte Management:

Fluid therapy is the foundation of initial stabilisation. Balanced isotonic crystalloids are commonly used to correct dehydration and improve tissue perfusion. Potassium supplementation may be required because total body potassium is often depleted, even when initial serum potassium appears normal. Phosphorus and magnesium should also be monitored and supplemented when indicated. As insulin drives potassium into cells, serum potassium may fall rapidly during treatment, making serial monitoring essential.

Monitoring During Therapy:

Close monitoring is essential throughout treatment. Blood glucose, BHB/ketones, electrolytes, hydration status, acid–base balance, urine output and clinical signs should be assessed serially. Blood glucose may be monitored using a portable blood glucose meter. Glucose monitoring can be stressful for patients and veterinary personnel. A novel continuous flash glucose monitoring system (FreeStyle Libre 2.0 Abbott®) eliminates the need for frequent capillary blood sampling (Eiermann *et al.*, 2024). The important point is that normalisation of blood glucose alone does not indicate resolution of DKA; improvement or resolution of ketosis, together with restoration of hydration and clinical stability, is essential before transition to routine diabetic management.

Complications:

DKA and its treatment can be associated with several complications, including hypoglycaemia, hypokalaemia, hypophosphatemia, hypomagnesaemia, persistent dehydration, fluid overload and rapid changes in osmolality. Severe electrolyte disturbances can contribute to weakness, neurological abnormalities and cardiac dysfunction. Careful monitoring and gradual correction of metabolic abnormalities help minimise these risks.

Prognosis:

The prognosis of DKA depends on the severity of metabolic disturbances, the presence of concurrent disease, the speed of diagnosis and the response to treatment. Patients that respond promptly to fluid therapy, insulin and correction of electrolyte abnormalities generally have a better chance of recovery. Identification and treatment of the underlying precipitating disease are also important for successful outcome.

Prevention and Client Education:

Prevention largely depends on consistent diabetic management and early recognition of deterioration. Owners should understand the importance of regular insulin administration, appropriate feeding, monitoring of water intake, appetite and clinical signs, and timely veterinary evaluation when vomiting, anorexia, lethargy or dehydration develops. Recording insulin doses and monitoring glucose or urine ketones, when advised, can help identify problems early.

Client education should also emphasise that missed insulin doses, concurrent illness and inadequate monitoring can increase the risk of metabolic decompensation. Good communication between the veterinary team and owner is therefore an important part of long-term diabetic care.

Conclusion:

DKA is not simply uncontrolled diabetes—it is a complex metabolic emergency. Early recognition, aggressive but carefully monitored stabilization, appropriate insulin therapy, correction of fluid and electrolyte abnormalities, nutritional support and identification of underlying disease are fundamental to successful management.

References:

- Cuddy, L. N., Hopper, K., Burkitt-Creedon, J. M., & Epstein, S. E. (2025). Retrospective evaluation of acid–base analysis in dogs and cats with diabetic ketosis (2017–2021): 96 cases. *Journal of Veterinary Emergency and Critical Care*, 35(1), 65-72.
- Eiermann, J. S., Hazuchova, K., Vidondo, B., Campos, M., Schuller, S., & Cui, Y. (2024). Comparison between a flash glucose monitoring system and a portable blood glucose meter for monitoring of cats with diabetic ketosis or ketoacidosis. *Animals*, 14(19), 2787.
- Gant, P., Barfield, D., & Florey, J. (2024). Comparison of insulin infusion protocols for management of canine and feline diabetic ketoacidosis. *Journal of Veterinary Emergency and Critical Care*, 34(1), 23-30.
- Hume, D. Z., Drobatz, K. J., & Hess, R. S. (2006). Outcome of dogs with diabetic ketoacidosis: 127 dogs (1993–2003). *Journal of veterinary internal medicine*, 20(3), 547-555.
- Hurrell, F. E., Drobatz, K. J., & Hess, R. S. (2016). Beta-hydroxybutyrate concentrations in dogs with acute pancreatitis and without diabetes mellitus. *Journal of veterinary internal medicine*, 30(3), 751-755.
- Silva, D. D., Cecci, G. R. M., Biz, G., Chiaro, F. N., & Zanutto, M. S. (2021). Evaluation of a flash glucose monitoring system in dogs with diabetic ketoacidosis. *Domestic animal endocrinology*, 74, 106525.
- Weingart, C., Lotz, F., & Kohn, B. (2012). Measurement of β -hydroxybutyrate in cats with nonketotic diabetes mellitus, diabetic ketosis, and diabetic ketoacidosis. *Journal of Veterinary Diagnostic Investigation*, 24(2), 295-300.