Emergency Endocrinology: brushing up on DKA

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Introduction

Diabetic ketoacidosis (DKA) is a **severe and potentially life-threatening complication of diabetes mellitus** characterized by the triad of **hyperglycemia, ketonemia (or ketonuria), and metabolic acidosis**. The condition requires urgent medical intervention and intensive monitoring. DKA develops when **absolute or relative insulin deficiency** occurs in combination with increased concentrations of counter-regulatory hormones such as glucagon, cortisol, growth hormone, and catecholamines.

Pathophysiology

The development of DKA involves complex metabolic alterations driven by **insulin deficiency and insulin resistance**. Concurrent diseases frequently trigger these hormonal disturbances and exacerbate metabolic dysregulation.

Common precipitating conditions include:

- pancreatitis
- bacterial urinary tract infection
- hyperadrenocorticism
- pyoderma
- neoplasia
- hepatic lipidosis or cholangiohepatitis
- chronic kidney disease
- viral infections (particularly in cats)

Insulin deficiency promotes **lipolysis and proteolysis**, resulting in increased circulating free fatty acids. These fatty acids are metabolized in the liver into ketone bodies, primarily **acetoacetate, acetone, and β-hydroxybutyrate**. Accumulation of these organic acids results in **metabolic acidosis**, which contributes to many of the clinical manifestations of the disease.

Clinical Manifestations

Clinical signs in patients with DKA reflect both **poorly controlled diabetes mellitus** and **systemic metabolic derangements**.

Typical signs include:

- polyuria and polydipsia
- weight loss despite polyphagia
- dehydration
- lethargy or depression
- vomiting and nausea
- diarrhea
- tachypnea or increased respiratory effort

In severe cases, patients may present with **shock, altered mentation, or profound dehydration**.

Diagnostic Criteria

The diagnosis of DKA is based on the presence of:

1. **Persistent hyperglycemia or glucosuria** indicating diabetes mellitus
2. **Ketosis**, detected as ketonemia or ketonuria
3. **Metabolic acidosis**, typically with reduced serum bicarbonate concentrations.

Hyperglycemia is present in nearly **all dogs with DKA at diagnosis (98–100%)** and can be detected using laboratory analyzers, point-of-care glucometers, or continuous glucose monitoring systems.
Ketonuria is detected in **94–100% of dogs**, although urine dipstick tests primarily measure **acetoacetate**, while **β-hydroxybutyrate is the predominant ketone body** in DKA. Quantitative measurement of β-hydroxybutyrate improves diagnostic accuracy.
Metabolic acidosis is confirmed by acid-base analysis and typically includes **decreased bicarbonate, increased base deficit, and an elevated anion gap**.

Further Diagnostic Workup

Because concurrent diseases frequently precipitate DKA, additional diagnostics are essential.

Recommended investigations include:

- complete blood count and serum biochemistry
- urinalysis with culture and sensitivity
- abdominal ultrasonography
- thoracic radiography
- cytology or fine-needle aspiration when neoplasia is suspected

Pancreatitis is commonly associated with DKA, although it remains unclear whether it is a cause or consequence of the disease. Diagnostic evaluation may include **abdominal ultrasonography and pancreatic lipase assays (cPL or fPL)**.

Concurrent endocrinopathies such as **hyperadrenocorticism in dogs or acromegaly in cats** may contribute to insulin resistance and should be investigated after initial stabilization.

Therapeutic Management

Stabilization and Fluid Therapy

Initial treatment focuses on **restoring circulating volume, correcting dehydration, and improving tissue perfusion**. Isotonic crystalloid fluids remain the cornerstone of therapy.
While **0.9% sodium chloride** was historically recommended, **balanced isotonic crystalloids** are increasingly preferred because excessive chloride may worsen metabolic acidosis.
Fluid therapy plans should include maintenance requirements, correction of dehydration, and replacement of ongoing losses.

Electrolyte Abnormalities

Electrolyte disturbances are common and must be corrected carefully.

Typical abnormalities include:

- **hypokalemia**, treated with potassium chloride supplementation
- **hypophosphatemia**, treated with potassium phosphate
- **hypomagnesemia**, treated with magnesium sulfate infusion

Correction of Metabolic Acidosis

In most patients, metabolic acidosis improves with **fluid therapy and insulin administration**. Sodium bicarbonate therapy remains controversial and should generally be reserved for **severe acidemia (pH < 7.0)**.

Insulin therapy is essential for:

- reducing hyperglycemia
- suppressing ketogenesis
- promoting utilization of glucose by peripheral tissues.

Rapid-acting crystalline insulin is typically used in the acute phase and may be administered as a **continuous rate infusion (CRI)** or **intermittent intramuscular injections**.
As blood glucose decreases, **dextrose supplementation** may be required to allow continued insulin administration until ketone metabolism has resolved.

Monitoring

Recommended monitoring includes:

- blood glucose every 1–2 hours
- serum electrolytes every 4–6 hours
- acid-base status every 4–6 hours
- ECG monitoring in cases of severe dyskalemia
- daily CBC and serum chemistry evaluation.

Assessment of hydration status, urine output, and body weight should also be performed regularly to guide fluid therapy adjustments.

Long-Term Management

After resolution of DKA, patients transition to **maintenance insulin therapy**, typically with intermediate-acting insulin preparations such as **lente or glargine**.

Long-term management focuses on:

- control of hyperglycemia
- appropriate nutrition and feeding schedules
- treatment of concurrent diseases
- prevention of insulin resistance
- monitoring for recurrent diabetic complications.

Prognosis

With appropriate intensive care, reported survival rates are approximately **70% in dogs and 61% in cats**.
Median hospitalization time is **five to six days**, although prognosis may be influenced by the presence of concurrent diseases and owner decisions regarding euthanasia.